A DISSERTATION ON EVALUATION OF MANNHEIM PERITONITIS INDEX TO PREDICT OUTCOME OF PATIENTS WITH PERITONITIS

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CERTIFICATE

This is to certify that this dissertation titled **"EVALUATION OF MANNHEIM PERITONITIS INDEX TO PREDICT OUTCOME OF PATIENTS WITH PERITONITIS"** has been prepared by **Dr.N.LAKSHMIPATHY** under my supervision in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu during the academic period 2008-2011 and is being submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University regulation for the award of the Degree of Master of surgery (M.S.General Surgery) and his dissertation is a bonafide work.

Prof.Dr.K.Kamaraj.M.S.,
Unit Chief,
Department of General Surgery,
Chengalpattu Medical College&
Hospital.

Prof. Dr.G.Raja Billy Graham.M.S
Head of the Department,
Department of General Surgery,
Chengalpattu Medical College&
Hospital.

Prof.Dr.P.Ramakrishnan.M.D.DLO.,
DEAN
Chengalpatu Medical College & Hospital,
Chengalpattu.

DECLARATION

I, **Dr.N.LAKSHMIPATHY**, solemnly declare that the dissertation **"EVALUATION OF MANNHEIM PERITONITIS INDEX TO PREDICT OUTCOME OF PATIENTS WITH PERITONITIS"** a bonafide work done by me in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu, Under the able guidance of Prof. **Dr.K.KAMARAJ.M.S**., Associate Professor, Chengalpattu Medical College, Chengalpattu.

Place: Chengalpattu.
Date:

(N. Lakshmipathy)

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CONTENTS

INTRODUCTION

Peritonitis is defined as inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. The peritoneum, which is an otherwise sterile environment, reacts to a variety of pathologic stimuli with a fairly uniform inflammatory response. Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (ie, chemical or mechanical).

Peritonitis is most often caused by introduction of an infection into the otherwise sterile peritoneal environment through organ perforation, but it may also result from other irritants, such as foreign bodies, bile from a perforated gall bladder or a lacerated liver, or gastric acid from a perforated ulcer. Women also experience localized peritonitis from an infected fallopian tube or a ruptured ovarian cyst. Patients may present with an acute or insidious onset of symptoms, limited and mild disease, or systemic and severe disease with septic shock.

Peritoneal infections are classified as primary (ie, from hematogenous dissemination, usually in the setting of immunocompromise), secondary (ie, related to a pathologic process in a visceral organ, such as perforation, trauma, or postoperative), or tertiary (ie, persistent or recurrent infection after adequate initial therapy).

Infections in the peritoneum are further divided into generalized (peritonitis) and localized (intra-abdominal abscess). This article focuses on the diagnosis and management of infectious peritonitis and abdominal abscesses. An abdominal abscess is seen in the images below. Reproducible scoring systems that allow a surgeon to determine the severity of the intrabdominal infection are essential to: 1) ratify the effectiveness of different treatment regimens, 2) scientifically compare surgical intensive care units, 3) help indicate individual risk to select patients who may require a more aggressive surgical approach and 4) be able to inform patient´s relatives with greater objectivity (6).

AIM OF STUDY

A study to confirm the predicative value of MPI among patients with intraoperative diagnosis of peritonitis at the surgical department , to evaluate severity of peritonitis and to make a prognosis of survival-mortality, considering the risk factors analyzed in this index

PATIENTS AND METHODS

A prospective, descriptive, transversal and observational study was undertaken. Patients included were all male and female patients, 14 years of age or older, seen at the Surgical Service . with diagnosis of peritonitis confirmed during surgery regardless of etiology. Once diagnosis of peritonitis had been determined by operative findings registered in the postoperative report, the patient was accepted into the study. Using data recollection sheets, risk factors found in MPI were classified according to values indicated in Table I and individual variable scores were added to establish initial MPI score. In addition to personal data such as name, age, sex, etc.,the following intrahospital information was registered: file number; dates of admission and discharge from the hospital; days hospitalized; date of surgery and information related to illness (surgical findings, medical treatment and evolution of illness). Patient evolution was followed, indicating presence of complications and discharge due to improvement or death. Time elapsed from initial diagnosis to moment of event (death or discharge from hospital) was determined. The minimum possible score was zero, if no adverse factor were present, and maximum was 47 if presence of all were confirmed. Patients were divided in three groups according to the following categories (MPI points) fewer than 15; from 16 to 25, and more than 25. . A life table, using the actuaria method, was constructed to compare patient survival with peritonitis severity according to MPI score. To determine significance of possible differences among three categories (< 15 points, between16 and 25, and >25 points).

REVIIEW OF LITERATURE

Untreated, acute peritonitis may be fatal. The fundamental role of operative therapy in the treatment of peritonitis was documented in 1926 when Kirschner reported that the mortality rate from intra-abdominal infections decreased from more than 90% to less than 40% during the period from 1890-1924 with the introduction of operative management. Other elements, such as advances in the understanding of damage control surgery, novel antibiotics, and improvements in intensive care unit (ICU) treatment have now reduced mortality to approximately 20%.The current approach to peritonitis and peritoneal abscesses targets correction of the underlying process, administration of systemic antibiotics, and supportive therapy to prevent or limit secondary complications due to organ system failure.

In 1986, Wacha H et al. published the Mannheim peritonitis index (MPI) based on analysis of 17 possible risks factors in patients with peritonitis; only eight factors were truly relevant to prognosis (age, sex, organ failure, cancer, duration of peritonitis, involvement of colon, extension of spread and character of peritoneal fluid) and were finally included in the index. The score considers clinic risk factors routinely found in preoperative and transoperative registers(14). This information is obtained during first laparotomy to establish an initial classification. Early

evaluation of severity of illness using MPI allows us to estimate the probability of patient survival(8,15). The MPI is one of the most simple scoring systems in use that allows the surgeon to easily determine outcome risk during initial surgery. The recollection of retrospective data is possible and valid, because MPI only requires information routinely found in surgical registers.

PERITONITIS AND ABDOMINAL SEPSIS

Frequency

The overall incidence of peritoneal infection and abscess is difficult to establish and varies with the underlying abdominal disease processes. The most common etiology of primary peritonitis is spontaneous bacterial peritonitis (SBP) caused by chronic liver disease. Up to 30% of all patients with liver cirrhosis with ascites develop SBP.The common etiologic entities of secondary peritonitis (SP) include perforated appendicitis; perforated gastric or duodenal ulcer; perforated (sigmoid) colon caused by diverticulitis, volvulus, or cancer; and strangulation of the small bowel (see Table 1). Necrotizing pancreatitis can also be associated with peritonitis in the case of infection of the necrotic tissue. The local inflammatory response in the study's patients with SP was significantly more severe than it was in patients with SBP, and the mortality rate during hospitalization was higher for SP than for SBP patients (66% vs 26.4%, respectively). However, patients with SP who underwent surgical treatment tended to have a lower mortality rate than did those who received only medical therapy (53.8% vs 81.8%, respectively). Among the surgically treated patients with SP, the survival rate was greater in those with the shortest time between diagnostic paracentesis and surgery (3.2+/-2.4 days in survivors vs 7.2+/-6.1 days in nonsurvivors, p=0.31).

Table 1. Common Causes of Secondary Peritonitis

Source Regions	**Causes**
Esophagus	Boerhaave syndrome Malignancy Trauma (mostly penetrating) Iatrogenic*
Stomach	Peptic ulcer perforation Malignancy (eg, adenocarcinoma, lymphoma, gastrointestinal stromal tumor) Trauma (mostly penetrating) Iatrogenic*
Duodenum	Peptic ulcer perforation Trauma (blunt and penetrating) Iatrogenic*
Biliary tract	Cholecystitis Stone perforation from gallbladder (ie, gallstone ileus) or common duct Malignancy Choledochal cyst (rare) Trauma (mostly penetrating) Iatrogenic*
Pancreas	Pancreatitis (eg, alcohol, drugs, gallstones) Trauma (blunt and penetrating) Iatrogenic*
Small bowel	Ischemic bowel Incarcerated hernia (internal and external) Closed loop obstruction Crohn disease Malignancy (rare) Meckel diverticulum Trauma (mostly penetrating)

Large bowel and appendix	Ischemic bowel Diverticulitis Malignancy Ulcerative colitis and Crohn disease Appendicitis Colonic volvulus Trauma (mostly penetrating) Iatrogenic
Uterus, salpinx, and ovaries	Pelvic inflammatory disease (eg, salpingo-oophoritis, tubo-ovarian abscess, ovarian cyst) Malignancy (rare) Trauma (uncommon)

The most common cause of postoperative peritonitis is anastomotic leak, with symptoms generally appearing around postoperative days 5-7. After elective abdominal operations for noninfectious etiologies, the incidence of SP (caused by anastomotic disruption, breakdown of enterotomy closures, or inadvertent bowel injury) should be less than 2%. Operations for inflammatory disease (ie, appendicitis, diverticulitis, cholecystitis) without perforation carry a risk of less than 10% for the development of SP and peritoneal abscess. This risk may rise to greater than 50% in gangrenous bowel disease and visceral perforation. .Peritonitis is also a frequent complication and significant limitation of peritoneal dialysis.]Peritonitis leads to increased hospitalization and mortality rates.

Etiology

Table 2. Microbiology of Primary, Secondary, and Tertiary Peritonitis

Peritonitis (Type)	Etiologic Organisms Class	Antibiotic Therapy (Suggested)
Primary	Gram-negative	Third-generation cephalosporin
Secondary	Gram-negative	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity Quinolones with anaerobic activity Quinolone and metronidazole Aminoglycoside and metronidazole
	Gram-positive	
	Anaerobic	
Tertiary	Gram-negative	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity Quinolones with anaerobic activity Quinolone and metronidazole Aminoglycoside and metronidazole Carbapenems Triazoles or amphotericin (considered in fungal etiology) (Alter therapy based on culture results.)
	Gram-positive	
	Fungal	

Pathophysiology

In peritonitis caused by bacteria, the physiologic response is determined by several factors, including the virulence of the contaminant, the size of the inoculum, the immune status and overall health of the host

(eg, APACHE II score), and the elements of the local environment, such as necrotic tissue, blood, or bile.Alterations in fibrinolysis (through increased plasminogen activator inhibitor activity) and the production of fibrin exudates have an important role in peritonitis. The production of fibrin exudates is an important part of the host defense, but large numbers of bacteria may be sequestered within the fibrin matrix. This may retard systemic dissemination of intraperitoneal infection and may decrease early mortality rates from sepsis, but it also is integral to the development of residual infection and abscess formation. As the fibrin matrix matures, the bacteria within are protected from host clearance mechanisms.The ultimate effect (containment vs persistent infection) of fibrin may be related to the degree of peritoneal bacterial contamination.

This bacterial load may locally overwhelm the host defense.Bacterial virulence factors[3]that interfere with phagocytosis and with neutrophil mediated bacterial killing mediate the persistence of infections and abscess formation. Among these virulence factors are capsule formation, facultative anaerobic growth, adhesion capabilities, and succinic acid production. Synergy between certain bacterial and fungal organisms may also play an important role in impairing the host's defense. One such synergy may exist between *B fragilis* and gram-negative bacteria, particularly *E coli,* where co-inoculation significantly increases bacterial proliferation and abscess formation.Enterococci may be important in enhancing the severity and persistence of peritoneal infections. ..

.Abscess formation occurs when the host defense is unable to eliminate the infecting agent and attempts to control the spread of this agent by compartmentalization. This process is aided by a combination of factors that share a common feature, ie, impairment of phagocytotic killing. The role of cytokines in mediation of the body's immune response and their role in the development of the systemic inflammatory response syndrome (SIRS) and multiple organ failure (MOF) have been a major focus of research over the past decade. Comparatively little data exist about the magnitude of the intraperitoneal/abscess cytokine response and implications for the host. Existing data suggest that bacterial peritonitis is associated with an immense intraperitoneal compartmentalized cytokine response. Higher levels of certain cytokines (ie, tumor necrosis factor-alpha [TNF-alpha], interleukin [IL]-6) have been associated with worse outcomes, as well as secondary (uncontrolled) activation of the systemic inflammatory cascade.

Presentation

The diagnosis of peritonitis is clinical. Abdominal pain, which may be acute or insidious, is the usual chief complaint. Initially, the pain may be dull and poorly localized (visceral peritoneum) and often progresses to steady, severe, and more localized pain (parietal peritoneum). If the underlying process is not contained, the

pain becomes diffuse. In certain disease entities (eg, gastric perforation, severe acute pancreatitis, intestinal ischemia), the abdominal pain may be generalized from the beginning. Anorexia and nausea are frequent symptoms and may precede the development of abdominal pain. Vomiting may be due to underlying visceral organ pathology (ie, obstruction) or be secondary to peritoneal irritation. On physical examination, patients with peritonitis generally appear unwell and in acute distress. Many of them have a temperature that exceeds 38° C, although patients with severe sepsis may become hypothermic. Tachycardia is caused by the release of inflammatory mediators, intravascular hypovolemia from anorexia vomiting and fever, and third-space losses into the peritoneal cavity. With progressive dehydration, patients may become hypotensive, as well as oliguric or anuric; with severe peritonitis, they may present in overt septic shock. On abdominal examination, almost all patients demonstrate tenderness to palpation. In most patients (even with generalized peritonitis and severe diffuse abdominal pain), the point of maximal tenderness or referred rebound tenderness roughly overlies the pathologic process (ie, the site of maximal peritoneal irritation).

Most patients demonstrate increased abdominal wall rigidity. The increase in abdominal wall muscular tone may be voluntary in response to or in anticipation of the abdominal examination or involuntary because of the peritoneal irritation.

Patients with severe peritonitis often avoid all motion and keep their hips flexed to relieve the abdominal wall tension. The abdomen is often distended, with hypoactive-to-absent bowel sounds. This finding reflects a generalized ileus and may not be present if the infection is well localized. Occasionally, the abdominal examination reveals an inflammatory mass. Rectal examination often elicits increased abdominal pain, particularly with inflammation of the pelvic organs, but rarely indicates a specific diagnosis. A tender inflammatory mass toward the right may indicate appendicitis, and anterior fullness and fluctuation may indicate a cul de sac abscess. In female patients, vaginal and bimanual examination findings may be consistent with pelvic inflammatory disease (eg, endometritis, salpingo-oophoritis, tubo-ovarian abscess), but exam findings are often difficult to interpret in severe peritonitis.

Indications

Early control of the septic source is mandatory and can be achieved by operative and nonoperative means. Operative management addresses the need to control the infectious source and to purge bacteria and toxins. The type and extent of surgery depends on the underlying disease process and the severity of intra-abdominal infection. Nonoperative interventions include percutaneous abscess drainage, as well as percutaneous and endoscopic stent placements. If an abscess is accessible for percutaneous drainage and if the underlying visceral organ pathology does not clearly

require operative intervention, percutaneous drainage is a safe and effective initial treatment approach.

Relevant Anatomy

The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac (ie, coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extraperitoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (ie, the peritoneal cavity).

The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.Normally, the amount of peritoneal fluid present is less than 50 mL, and only small volumes are transferred across the considerable surface area in a steady state each day. The peritoneal fluid represents a plasma ultrafiltrate, with electrolyte and

solute concentrations similar to that of neighboring interstitial spaces and a protein content of less than 30 g/L, mainly albumin. In addition, peritoneal fluid contains small numbers of desquamated mesothelial cells and various numbers and morphologies of migrating immune cells (reference range is <300 cells/µL, predominantly of mononuclear morphology)The peritoneal cavity is divided incompletely into compartments by the mesenteric attachments and secondary retroperitonealization of certain visceral organs. A large peritoneal fold, the greater omentum, extends from the greater curvature of the stomach and the inferior aspect of the proximal duodenum downward over a variable distance to fold upon itself (with fusion of the adjacent layers) and ascends back to the taenia omentalis of the transverse colon. This peritoneal fold demonstrates a slightly different microscopic anatomy, with fenestrated surface epithelium and a large number of adipocytes, lymphocytes, and macrophages, and it functions as a fat storage location and a mobile immune organ. The compartmentalization of the peritoneal cavity, in conjunction with the greater omentum, influences the localization and spread of peritoneal inflammation and infections.

Workup

Laboratory Studies

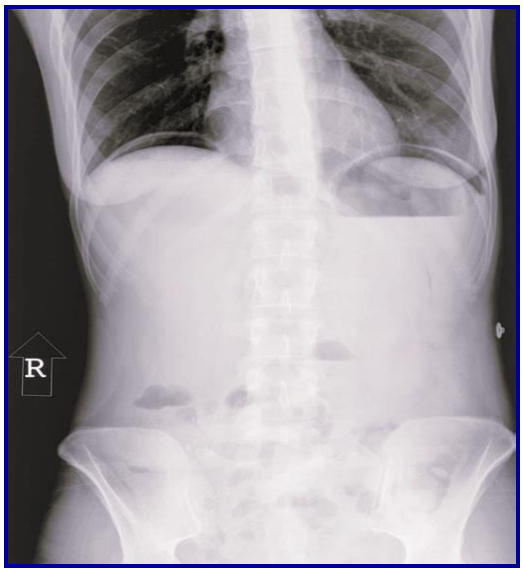
- CBC with differential - Most patients will have leukocytosis (>11,000 cells/µL),

- Blood chemistry - May reveal dehydration and acidosis
- PT, PTT, and INR
- Liver function tests - If clinically indicated
- Amylase and lipase - If pancreatitis is suspected
- Urinalysis (UA) - To rule out urinary tract diseases (eg, pyelonephritis, renal stone disease); however, patients with lower abdominal and pelvic infections often demonstrate WBCs in the urine and microhematuria.
- Stool sample - In patients with diarrhea, evaluate a stool sample — employing a *Clostridium difficile* toxin assay, a WBC count, and a specific culture (ie, *Salmonella, Shigella,* cytomegalovirus [CMV]) — if the patient's history suggests infectious enterocolitis.
- Aerobic and anaerobic blood cultures
- Peritoneal fluid (ie, paracentesis, aspiration of abdominal fluid collections, intraoperative peritoneal fluid cultures)
    - Diagnostic peritoneal lavage (DPL) may be helpful in patients who do not have conclusive signs on physical examination or who cannot provide an adequate history. A DPL with more than 500 leukocytes/mL is considered positive and suggests peritonitis.

- Evaluate the sample for pH, glucose, protein, lactate dehydrogenase (LDH), cell count, Gram stain, and aerobic and anaerobic cultures.
- Include analysis if pancreatitis or pancreatic leak is suspected.
- Test for bilirubin when a biliary leak is suspected and for fluid creatinine level when a urinary leak is suspected.
- Compare the peritoneal levels to the respective serum level

Imaging Studies

- Radiographs
    - Plain films of the abdomen (eg, supine, upright, and lateral decubitus positions) are often the first imaging studies obtained in patients presenting with peritonitis. Their value in reaching a specific diagnosis is limited.
    - Free air is present in most cases of anterior gastric and duodenal perforation but is much less frequent with perforations of the small bowel and colon and is unusual with appendiceal perforation. ..



AIR UNDER DIAPHARAGM

MULTPLE AIR FLUID LEVELS I N INTESTINAL OBSTRUCTION

COFFEE BEAN APPEARENCE IN SIGMOID VOLVULUS

- Ultrasonography
    - Abdominal ultrasonography may be helpful in the evaluation of right upper quadrant (eg, perihepatic abscess, cholecystitis, biloma, pancreatitis, pancreatic pseudocyst), right lower quadrant, and pelvic pathology (eg, appendicitis, tubo-ovarian abscess, Douglas pouch abscess), but the examination is sometimes limited because of patient discomfort, abdominal distension, and bowel gas interference.
    - Ultrasonography may detect increased amounts of peritoneal fluid (ascites), but its ability to detect quantities of less than 100 mL is limited. .

- Ultrasonographically guided aspiration and placement of drains has evolved into a valuable tool in the diagnosis and treatment of abdominal fluid collections (see Medical Therapy).

- CT scanning

  - If the diagnosis of peritonitis is made clinically, a CT scan is not necessary and generally delays surgical intervention without offering clinical advantage. CT scans of the abdomen and pelvis remain the diagnostic study of choice for peritoneal abscess and related visceral pathology. CT scanning is indicated in all cases in which the diagnosis cannot be established on clinical grounds and findings on abdominal plain films. Whenever possible, the CT scan should be performed with enteral and intravenous contrast. CT scans can detect small quantities of fluid, areas of inflammation, and other GI tract pathology, with sensitivities that approach 100%.

  - Peritoneal abscesses and other fluid collections may be aspirated for diagnosis and drained under CT guidance; this technique has become a mainstay of therapy (see Medical Therapy).

MULTIPLE AIR FLUID LEVELS IN CT

- Nuclear medicine scans
- .. They are most frequently used in the evaluation of fever of unknown origin or in patients with persistent fever despite adequate antibiotic treatment and negative CT scan findings.

- Magnetic resonance imaging (MRI)
  - MRI is an emerging imaging modality for the diagnosis of suspected intra-abdominal abscesses. .

- Contrast studies
  - Conventional contrast studies (ie, Gastrografin swallow, upper GI tract study with follow-through, colorectal contrast enema, fistulogram, contrast studies of drains and stents) are reserved for specific indications in the setting of suspected peritonitis or peritoneal abscess.

Diagnostic Procedures

- See Surgical Therapy for a discussion of laparoscopy.

Treatment

Medical Therapy

The general principles guiding the treatment of intra-abdominal infections are 4-fold: (1) to control the infectious source, (2) to eliminate bacteria and toxins, (3) to maintain organ system function, and (4) to control the inflammatory process. The treatment of peritonitis is multidisciplinary, with complimentary application of medical, operative and nonoperative interventions included in the therapy. Medical support includes (1) systemic antibiotic therapy; (2) intensive care with hemodynamic, pulmonary, and renal support; (3) nutrition and metabolic support; and (4) inflammatory response modulation therapy. Early control of the septic source is mandatory and can be achieved by operative and nonoperative means. Nonoperative interventional therapies include percutaneous drainage of abscesses and percutaneous and endoscopic stent placements. Treatment of peritonitis and intra-abdominal sepsis always begins with volume resuscitation, correction of potential electrolyte and coagulation abnormalities, and empiric broad-spectrum parenteral antibiotic coverage.

Antibiotic therapy

Spontaneous bacterial peritonitis

Untreated SBP has a mortality rate of up to 50%, but with prompt diagnosis and treatment of the condition, this figure may be reduced to 20%. Empiric therapy with a third-generation cephalosporin must begin promptly and can subsequently be narrowed according to the culture results. Avoid aminoglycosides in patients with liver disease, because these patients are at an increased risk for nephrotoxicity.

The optimal duration of therapy is not known; traditionally, a course of 10 days is recommended, although studies have suggested that 5 days of therapy (with documentation of a decrease of peritoneal fluid WBC count to <250 cells/μ L) may be sufficient in most cases.

Secondary and tertiary peritonitis

In secondary and tertiary peritonitis, systemic antibiotic therapy is the second mainstay of treatment.[6]Several studies suggest that antibiotic therapy is not as effective in the infection's later stages and that early (preoperative) systemic antibiotic therapy can significantly reduce the concentration and growth rates of viable bacteria in the peritoneal fluid. Antibiotic therapy begins with empiric coverage (effective against common gram negative and anaerobic pathogens) and should be initiated as soon as possible, with a transition made to narrower spectrum agents as culture results become available.

Perforations of upper GI tract organs are associated with gram-positive bacteria, whereas the distal small bowel and colon perforations involve polymicrobial aerobic and anaerobic species. Most studies suggest that single-drug therapy is as effective as dual or triple combination therapy in mild to moderate abdominal infections.In severe and hospital-acquired intra-abdominal infections, imipenem, piperacillin/tazobactam, and a combination of aminoglycosides and metronidazole are often effective.

The optimal duration of antibiotic therapy must be individualized and depends on the underlying pathology, severity of infection, speed and effectiveness of source control, and patient response to therapy. In uncomplicated peritonitis in which there is early, adequate source control, a course of 5-7 days of antibiotic therapy is adequate in most cases. Mild cases (eg, early appendicitis, cholecystitis) may not need more than 24-72 hours of postoperative therapy. Inadequate initial therapy has been linked to worse outcomes, and these outcomes could not be significantly changed by later specific or prolonged therapy. Antimicrobial therapy should continue until signs of infection (eg, fever, leukocytosis) have resolved; when signs of infection continue, persistent infection or the presence of a nosocomial infection should be investigated.

Some patients demonstrate persistent signs of inflammation without a defined infectious focus. In these patients, continued broad-spectrum antibiotic therapy may be more harmful than beneficial (eg, emergence of resistant organisms, *C difficile* colitis), and a trial of antibiotic therapy

cessation under close surveillance may be warranted.Of note, antibiotics alone are seldom sufficient to treat intra-abdominal abscesses, and adequate drainage of the abscess is of paramount importance. For most of the commonly used antibiotics, abscess fluid antibiotic levels are generally below the minimum inhibitory concentration-90 (MIC90) for *B fragilis* and *E coli,* and repeated dosing or high-dose therapy does not improve penetration significantly.

Nonoperative drainage

CT scan – and ultrasonographically guided percutaneous drainage are well established as effective source controls and may in some cases decrease the need for surgical therapy. In some instances, success also includes the ability to delay surgery until the acute process and sepsis are resolved and a definitive procedure can be performed under elective circumstances.

Percutaneous and surgical drainage should not be considered competitive but rather complementary. If an abscess is accessible to percutaneous drainage and the underlying visceral organ pathology does not clearly require an operative approach, percutaneous drainage can be used safely and effectively as the primary treatment modality. In these cases, patients must be closely monitored, and improvement should be observed in less than 24-48 hours. With lack of improvement, patients

must be reevaluated aggressively (eg, repeat CT scan) and the therapeutic strategy should be altered accordingly.

Surgical Therapy

Surgery remains a cornerstone of treating peritonitis. Any operation should address the first 2 principles of the treatment of intra-peritoneal infections: early and definitive source control and elimination of bacteria and toxins from the abdominal cavity. The issue of timing and adequacy of surgical source control is paramount because an improper, untimely, or incorrect operation may have an overwhelmingly negative effect on outcome (compared to medical therapy).

The operative approach is directed by the underlying disease process and the type and severity of the intra-abdominal infection. In many cases, the indication for operative intervention will be clear, as in cases of peritonitis caused by ischemic colitis, a ruptured appendix, or colonic diverticula. The surgeon should always strive to arrive at a specific diagnosis and delineate the intra-abdominal anatomy as accurately as possible prior to the operation.

However, in severe abdominal sepsis, delays in operative management may lead to a significantly higher need for reoperations and to worse outcomes overall; early exploration (ie, prior to completion of diagnostic studies) may be indicated. Surgical intervention may include resection of a perforated viscus with re-anastomosis or creation of a fistula.

To reduce the bacterial load, a lavage of the abdominal cavity is performed, with particular attention to areas prone to abscess formation (e.g., paracolic gutters, subphrenic area).

These patients may best be served by a period of 12-24 hours of observation and intensive medical support. Deterioration of the patient's clinical status or development of organ-specific indications (eg, intra-abdominal bleed, gas-forming infection of the pancreas) should lead to prompt operation. Percutaneous treatment is reserved for the management of defined peripancreatic fluid collections in stable patients. Pancreatic abscess or infected pancreatic necrosis generally should be treated with surgical debridement and repeated exploration. If an anastomotic dehiscence is suspected, percutaneous drainage is of limited value, and the patient should be treated surgically.

Open-abdomen technique and scheduled reoperation

In certain situations, staging the operative approach to intraperitoneal infections is appropriate. Staging may be performed as a scheduled second-look operation or through open management, with or without temporary closure (eg, mesh, VAC technique).

Second-look operations may be used in a damage control fashion. In these cases, the patient at initial operation is severely ill and unstable from septic shock or coagulopathy (eg, mediator liberation, disseminated intravascular coagulation). The goal of the initial operation is to provide

preliminary drainage and to remove obviously necrotic tissue. Then, the patient is resuscitated and stabilized in an ICU setting for 24-36 hours and returned to the operating room for a more definitive drainage and source control.

In conditions related to bowel ischemia, the initial operation aims to remove all frankly devitalized bowel. The second-look operation serves to reevaluate for further demarcation and decision-making regarding reanastomosis or diversion.In severe peritonitis, particularly with extensive retroperitoneal involvement (eg, necrotizing pancreatitis), open treatment with repeat reexploration, debridement, and intraperitoneal lavage has been shown to be effective.

Laparoscopy

Laparoscopy is gaining wider acceptance in the diagnosis and treatment of abdominal infections. As with all indications for laparoscopic surgery, outcomes vary depending on the skill and experience of the laparoscopic surgeon.

Initial laparoscopic examination of the abdomen can assist in determination of the etiology of peritonitis (eg, right lower quadrant pathology in female patients). Laparoscopic surgery is commonly used in the treatment of uncomplicated appendicitis, although in preliminary studies, outcomes for complicated appendicitis have generally been positive. For complicated and uncomplicated appendicitis, the laparoscopic

approach is associated with a shorter length of stay and fewer wound infections than the open approach; however laparoscopic surgery may be associated with a higher rate of intra-abdominal abscess.

Laparoscopic diagnosis and peritoneal lavage in patients with peritonitis secondary to diverticulitis has been shown to be safe and has helped to avoid the need for colostomy in many patients in small clinical trials. Successful laparoscopic repair of perforated gastric and duodenal ulcers has also been reported.

The treatment of perihepatic infections via laparoscopic approach has been well established in acute cholecystitis, where laparoscopic cholecystectomy has become the mainstay of therapy. More recently, primary treatment of subphrenic abscesses and laparoscopic, ultrasonographically assisted drainage of pyogenic liver abscesses have been performed successfully.

Individual reports also describe successful drainage of peripancreatic fluid collections and complicated intra-abdominal abscesses that are not amenable to CT scan – or ultrasonographically guided percutaneous drainage.

As minimally invasive procedures continue to advance technologically, use of these approaches is likely to increase, reducing the need for the open surgical approach for peritoneal abscess drainage.

Preoperative Details:

Volume resuscitation and prevention of secondary organ system dysfunction are of utmost importance in the treatment of patients with intra-abdominal infections. Depending on the severity of the disease, these patients should have Foley catheters placed to monitor urine output. Use invasive hemodynamic monitoring in severely ill patients to guide volume resuscitation and inotropic support. Correct existing serum electrolyte disturbances and coagulation abnormalities as best as possible before any intervention. Begin empiric broad-spectrum systemic antibiotic therapy as soon as the diagnosis of intra-abdominal infection is suspected and tailor therapy according to the underlying disease process and culture results. Remember that patients with peritonitis often have severe abdominal pain. Provide adequate analgesia with parenteral narcotic agents as soon as possible. In the setting of significant nausea, vomiting, or abdominal distension caused by obstruction or ileus, institute nasogastric decompression as soon as possible. Consider intubation and ventilator support early in patients with evidence of septic shock or altered mental status to prevent further decompensation.

Even if patients do not appear critically ill initially, arranging for postoperative intensive care support before the operation is often wise, particularly in patients of advanced age and those with significant comorbidities.In patients with severe infections and certain disease processes (eg, necrotizing pancreatitis, bowel ischemia), informed consent

should include the potential need for several reoperations and enteric diversion. The involved physicians and surgeon should not downplay the significant morbidities associated with abdominal sepsis when discussing these issues with the patient and/or family.

Intraoperative Details

The goals of operative treatment of peritonitis are to eliminate the source of contamination, to reduce the bacterial inoculum, and to prevent recurrent or persistent sepsis. A vertical midline incision is the incision of choice in most patients with generalized peritonitis because it allows access to the entire peritoneal cavity. In patients with localized peritonitis (eg, acute appendicitis, cholecystitis), an incision directly over the site of pathology (eg, right lower quadrant, right subcostal) is usually adequate. In patients with an unclear etiology of the peritonitis, initial diagnostic laparoscopy may be useful.The intra-abdominal anatomy may be significantly distorted because of inflammatory masses and adhesions. Normal tissue planes and boundaries may be obliterated. The inflamed organs are often very friable, and the surgeon must exercise great caution when exploring the patient with peritoneal infection.Hemodynamic instability may occur at any time during treatment because of bacteremia and cytokine release. Patients often demonstrate significant fluid shifts with third spacing. Swelling of the bowel, retroperitoneum, and abdominal wall may preclude safe abdominal closure after prolonged cases in patients who are severely ill.Inflammation causes regional hyperemia, and sepsis

may cause coagulation deficits and platelet dysfunction, leading to increased bleeding. Careful dissection and meticulous hemostasis are of utmost importance.

When faced with extensive abdominal inflammatory disease and septic shock, draining the infection temporarily, controlling the visceral leak quickly (eg, oversewing, enteric diversion), and deferring any definitive repair until after the patient has recovered from the initial insult (ie, damage control operation) may be better.One of the critical decisions in the surgical treatment of patients with severe peritonitis is regarding whether to use a closed-abdomen or open-abdomen technique. The goal of the closed-abdomen technique is to provide definitive surgical treatment at the initial operation; perform primary fascial closure and perform repeat laparotomy only when clinically indicated. The goal of the open-abdomen technique is to provide easy direct access to the affected area. Source control is achieved through repeated reoperations or open packing of the abdomen. This technique may be well suited for initial damage control in extensive peritonitis.Also consider patients who are at high risk for development of abdominal compartment syndrome (eg, intestinal distension, extensive abdominal wall and intra-abdominal organ edema) for this technique because attempts to perform primary fascial closure under significant tension in these circumstances are associated with an increased incidence of MOF (eg, renal, respiratory), necrotizing abdominal wall infections, and mortality.

Postoperative Details

Postoperatively, monitor all patients closely in the appropriate clinical setting for adequacy of volume resuscitation, resolution or persistence of sepsis, and the development of organ system failure. Appropriate systemic broad-spectrum antibiotic coverage must be continued without interruption for the appropriate time (see Medical Therapy). The patient's overall condition should improve significantly and progressively within 24-72 hours of the initial treatment (ie, resolution of the signs and symptoms of infection, mobilization of interstitial fluid). This time course may be prolonged in patients who are critically ill with significant multiple organ system dysfunction. A lack of improvement should prompt an aggressive search for a persistent or recurrent intraperitoneal or new extraperitoneal infectious focus.Patients requiring surgical intervention for peritonitis demonstrate a significantly increased risk for surgical site infections and wound healing failure; monitor patients closely for this potential complication. All patients who are critically ill and patients receiving prolonged antibiotic therapy are at increased risk for developing secondary opportunistic infections (eg, *C difficile* colitis, fungal infections, central venous catheter infections, ventilator-associated pneumonia); monitor patients closely for signs and symptoms of these complications.

Nutrition

In general, patients with peritonitis develop some degree of gut dysfunction (eg, ileus) after exploration. Consider establishing some form of nutritional support early in the course of treatment because most patients have an insufficient enteral intake for a variable amount of time preoperatively. The existing data support that enteral nutrition is superior to parenteral hyperalimentation. If enteral feeding is contraindicated or not tolerated, parenteral nutrition should be instituted.

Follow-up

After resolution of peritonitis and peritoneal abscesses, follow-up care is directed mostly by specifics of the underlying disease process and the presence or absence of chronic complications (eg, enterocutaneous fistulae). Patients with simple peritoneal infections after appendicitis or cholecystitis are usually cured and do not require long-term follow-up care. Patients with peritoneal operations for perforated peptic ulcer disease, Crohn disease, pancreatitis, and others often require lifelong medical therapy and treatment of recurrent complications.

Complications

Surgical site infection/dehiscence

The incidence of surgical site infection increases with the degree of contamination; therefore, surgical site infection occurs at much higher rates after operations for peritonitis and peritoneal abscess (ie, 5-15%

compared to <5% for elective abdominal operations for noninfectious etiologies). Surgical site infection may be expected if the wound is closed in the setting of gross abdominal contamination Perioperative systemic antibiotics, the use of wound protector devices, and lavage of the wound at the end of therapy do not reliably prevent this complication. These wounds should be left open and be treated with wet-to-dry dressing changes several times a day or VAC dressing should be applied.

Impaired wound healing

Complications related to percutaneous drainage

Percutaneous drainage procedures carry a risk of related significant complications of less than 10% (range 5-27%) depending on the underlying pathology and abscess location. These complications include bleeding, injury, erosion, transgression of small and large bowel, fistula formation, and others. Strategies to prevent these problems include correction of coagulation problems and determination of the exact etiology, location, and anatomic relationships of the abscess. Indication for percutaneous treatment of complex abscesses and patients with a persistent enteric leak should be reviewed critically, and operative treatment should not be delayed with lack of adequate patient improvement.

Tertiary peritonitis

Persistence of intra-abdominal infection (ie, tertiary peritonitis) is a complication that may occur following the treatment of primary or secondary peritonitis and peritoneal abscess..

Complications related to the open-abdomen technique

One of the complications related to treatment of severe intra-abdominal infections with the open-abdomen technique and multiple reoperations is the development of enterocutaneous fistulae. .

High-output and proximal fistulae often require a delayed surgical repair. Optimal timing of this repair is critical. Initial inflammatory adhesions and dense scar formation may make safe reexploration impossible. Maturation of the scar tissue occurs over 6-12 months. Close observation of the patient's overall condition and nutritional status is important during that time. Deterioration of the patient's condition may force an earlier reoperation.

Complications related to abdominal compartment syndrome

ACS is a well-recognized disease entity related to acutely increased abdominal pressure (ie, intra-abdominal hypertension [IAH]) and is associated with the development of multiple organ dysfunction.

Complications related to enteric insufficiency

Extensive initial (gastrointestinal) disease, chronic recurrent infections, and associated reoperations may lead to enteric insufficiency because of short gut, pancreatic insufficiency, or hepatic dysfunction. Treatment of these problems can be quite challenging and can require a

multispecialty approach to optimize gastrointestinal function and nutritional status.

Outcome and Prognosis

Spontaneous bacterial peritonitis

The overall mortality rate of patients with SBP may exceed 30% if diagnosis and treatment are delayed, but the mortality rate is less than 10% in fairly well-compensated patients with early therapy. As many as 70% of patients who survive an episode of SBP have a recurrent episode within 1 year, and, for these patients, the mortality rate approaches 50%. Some studies suggest that the recurrence rate of SBP may be decreased to less than 20% with long-term antibiotic prophylaxis (eg, quinolones, trimethoprim-sulfamethoxazole); however, whether this improves long-term survival without liver transplantation is unclear.

Secondary peritonitis and peritoneal abscess

Treatment success of peritoneal infections is defined as adequate source control with resolution of sepsis and clearance of all residual intra-abdominal infection. With percutaneous treatment, the definition of success includes the avoidance of further operative intervention and, in some cases, the delay of surgery until after resolution of the initial sepsis. Over the past decade, the combination of better antibiotic therapy, more aggressive intensive care, and earlier diagnosis and therapy with a combination of operative and percutaneous techniques have led to a

significant reduction in morbidity and mortality related to intra-abdominal sepsis.Uncomplicated SP and simple abscesses carry a mortality rate of less than 5%, but this rate may increase to greater than 30-50% in severe infections. The overall mortality rate related to intra-abdominal abscess formation is less than 10-20%. Factors that independently predict worse outcomes include advanced age, malnutrition, presence of cancer, a high APACHE II score on presentation, preoperative organ dysfunction, the presence of complex abscesses, and failure to improve in less than 24-72 hours after adequate therapy.

In severe intra-abdominal infections and peritonitis, the mortality rate may increase to greater than 30-50%. The concurrent development of sepsis, SIRS, and MOF can increase the mortality rate to greater than 70%, and, in these patients, more than 80% of deaths occur with an active infection present.Several scoring systems (eg, APACHE II, SIRS, multiple organ dysfunction syndrome [MODS], Mannheim peritonitis index) have been developed to assess the clinical prognosis of patients with peritonitis. Most of these scores rely on certain host criteria, systemic signs of sepsis, and complications related to organ failure. Although valuable for comparing patient cohorts and institutions, these scores have limited value in the specific day-to-day clinical decision-making process for any given patient. In general, the mortality rate is less than 5% with an APACHE II of less than 15 and rises to greater than 40% with scores above 15. Rising APACHE II scores on days 3 and 7 are associated with an increase of

mortality rates to greater than 90%, whereas falling scores predict mortality rates of less than 20%.

The mortality rate without organ failure generally is less than 5% but may rise to greater than 90% with quadruple organ failure. A delay of more than 2-4 days of either medical therapy or surgical therapy has been clearly associated with increased complication rates, the development of tertiary peritonitis, the need for reoperation, multiple organ system dysfunction, and death.

Outcomes are worse in patients requiring emergent reoperations for persistent or recurrent infections (30-50% increase in the mortality rate); however, patients undergoing early planned second-look operations do not demonstrate this trend.

Persistent infection, recovery of enterococci, and multidrug-resistant gram-negative organisms, as well as fungal infection, are related to worse outcomes and recurrent complications.

Patients older than 65 years have a 3-fold increased risk of developing generalized peritonitis and sepsis from gangrenous or perforated appendicitis and perforated diverticulitis than younger patients and are 3 times more likely to die from these disease processes. Older patients with perforated diverticulitis are 3 times more likely than younger patients to have generalized rather than localized (ie, pericolic, pelvic) peritonitis. These findings are consistent with the hypothesis that the

biologic features of peritonitis differ in elderly persons, who are more likely to present with an advanced or more severe process than younger patients with peritonitis.

Overall, studies suggest that host-related factors are more significant than the type and source of infection with regard to the prognosis in intra-abdominal infections.

Table 1 Mannheim Peritonitis Index Scoring

Risk Factor	Weighting if present
Age>50 Years	5
Female sex	5
Organ failure	7
Mailignancy	4
Preoperative duration of peritonitis>24hrs	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudate Clear Cloudy, Purulent Fecal	 0 6 12

Future and Controversies

Laparoscopy is gaining wider acceptance in the diagnosis and treatment of abdominal infections (see Laparoscopy in Surgical Therapy). However, no definitive guidelines have been established regarding the optimal selection of patients for successful laparoscopic repair. As minimally invasive procedures continue to advance technologically, use of these approaches is likely to increase, reducing the need for the open surgical approach for peritoneal abscess drainage.

RESULTS

From Jan 2009 to Nov 30 2010 of about 150 patients with peritonitis confirmed during surgical intervention were admitted to the Surgical Service,except one patient treated conservatively by bilateral flank drain due to Ca cervix on radiotherapy.

Study Group General Data

Of the sample of 150 patients, 28 were female (18.7%) and 122were male (81.3%). Group mean age was 41.8 years with a median of 40 years and a range from 14 years and above. Mean age of survivors was 39.78 years of age (± 13.8); among non-survivors, mean age was 53 years (± 11.3) (p <0.0001).

Of the 150 patients 149 were operated and one was bilateral flank drain , 23(15.3%) died (global mortality 6%),17 (11.4%) were wound sepsis and 110(73.3%) were discharged without complication. Origin of peritonitis was from more than 6 different anatomic sites and was due to various causes (Table Chart-1.1-1.3).

Table . Peritonitis assigned to Anatomical location

	Died	**Discharged**	**Wound sepsis & Discharged**	**Total**
Duodenal Perforation	7 (8%)	67 (81%)	9 (11%)	83
Gastric Perforation	6 (38%)	7 (44%)	3 (18%)	16
Appendicular Pathology	1 (7%)	12 (80%)	2 (13%)	15
Small bowel	5 (26%)	13 (68%)	1 (6%)	19
Large Bowel	2 (29%)	5 (71%)	0	7
Miscellaneous	2 (20%)	6 (60%)	2 (20%)	10

Mannheim Criteria Data

Group mean MPI score was 18 points. Among surviving patients, mean score was 16 points and among non-survivors, mean was 27 points ($p < 0.0001$). We can observe the study group life table. survival curves of the three subgroups (<15 , 16–25 and >25) have differences that are statistically significant. succinctly, break down information of each risk factor according to the following categories: a) MPI scores<15, 16–25 and >25, Results of Odds ratio for each risk factor, for age over 50 years is 5.346 and Gender is 0.(Chart-2.1 to 5.1)

DISCUSSION AND CONCLUSION

A glance at the life chart shows a difference in prognosis of the three established intervals. There is 1%of deaths in patients with scores <15 MPI points, and survival of patients with interval of 15–25 points was superior to those with 25 points, confirming the predictive value of MPI among patients with surgically diagnosed peritonitis . This implies that survival differences observed among three intervals (<15, 16–25, and >25 MPI points) are statistically significant(p = <0.001). Mean MPI score of all patients with peritonitis studied was 18 points. Among patients who died, mean was 27 points compared to 16 points among survivors. This difference is statistically significant (p = <0.0001). Other foreign studies show mean MPI scores between 19 and 34 points (range 0to47points)(6,15,18,21,39,42-45). MPI among survivors and non-survivors reported is 25 and 31 MPI points, respectively(46). In our study, overall mortality rate was 15.3%; other studies report global mortality rates from 3.9% to 54% (6,14,15,18,20,21,23,26,32,34, 42,43, 45,47,53).

In concordance with the life table, when MPI score increased, mortality increased, which coincides with other publications.49 .MPI is an important index for predicting patient outcome in peritonitis(54). Variouspublications use more than one score study group outcome in patients with peritonitis, surgically In the Mexican study previously cited, was concluded that the only prognostic factor with statistic value was MPI

score in patients with abdominal sepsis; however, combined with APACHE II, prognosis was more thorough, realistic, and significant(47).

Others differ, concluding that sensitivity and specificity with MPI is greater than that calculated with APACHE II(37). When comparing risk factors of each variable indicating the presence or absence of adverse factor among survivors and non-survivors, a mirror image is expected, as occurred. This means that adverse factor is low and favorable factor is high in survivors, and the contrary in non-survivors When considering each risk factor, constructing a contingency table in which presence or absence of adverse factor and result (death or survival) are considered, OR value obtained allows us to weigh, each risk factor as follows: presence of malignancy; age 50 years; generalized peritonitis; presence of fecal peritoneal fluid, and female gender. Contrary to what was expected, in this study colonic origin was not an adverse factor. Even though mortality rate in presence of malignancy is high, the result was not conclusive due to the small number of patients with malignancy in this series.(Chart-2.1to5.3)

Mean age of survivors was 39.78 years (SD ±13.8); mean age among non-survivors was 53 years (SD ±11.3) considerably younger than other studies using MPI in which mean age was 49–66 years old (range 2 to 93 years). Although our study group excluded pediatric patients, our mean age was younger than other series that included children (6,16,26, 28, 42,43,53). This can be due to difference in population pyramids.

Generalized peritonitis with early intervention ie less than 24hrs have better prognosis and intervention more than 24hrs and non colonic origin has poor prognosis and long duration of hospital stay average hospital stay was 12.96 days ranging from 0 to 45 days. (Chart -4)

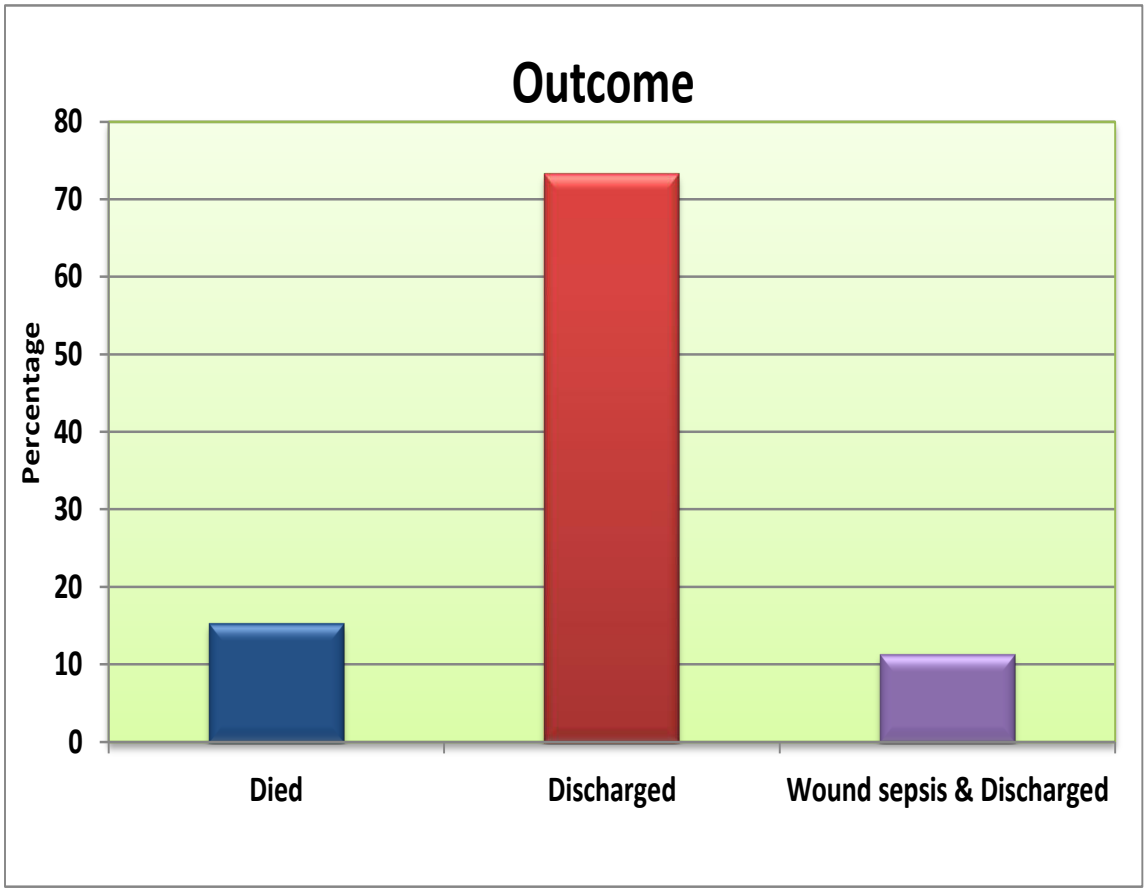
Approximately 18.7% were female and 81.3% were male with mortality of 18%(5/28)and 15%(18/122) respectively. Noncolonic origin is also considered an adverse factor with 95% mortality when compared to colonic origin 5% To conclude that MPI is a useful method to determine study group outcome in patients with peritonitis surgically evaluated.(Chart-5.3) Considering survival related with character of peritoneal fluid we found :clear fluid had mortality 2.2% (2/90) ,purulent fluid mortality rate of 32.7% (19/58),and fecal fluid had mortality 100%(2/2).

To conclude that MPI is a useful method to determine study group outcome in patients with peritonitis surgically evaluated. All MPI adverse factors, except colonic origin, behaved as expected, and the following were especially useful: presence of the organic failure; time elapsed >24 h; presence of malignity; age >50 years, and generalized extension of peritonitis. MPI, together with surgeon's clinical judgment of each case, may be another possible use of this score, aiding the surgeon in making the always difficult decision of reintervention in a patient.

CHARTS

Outcome –Chart1.1

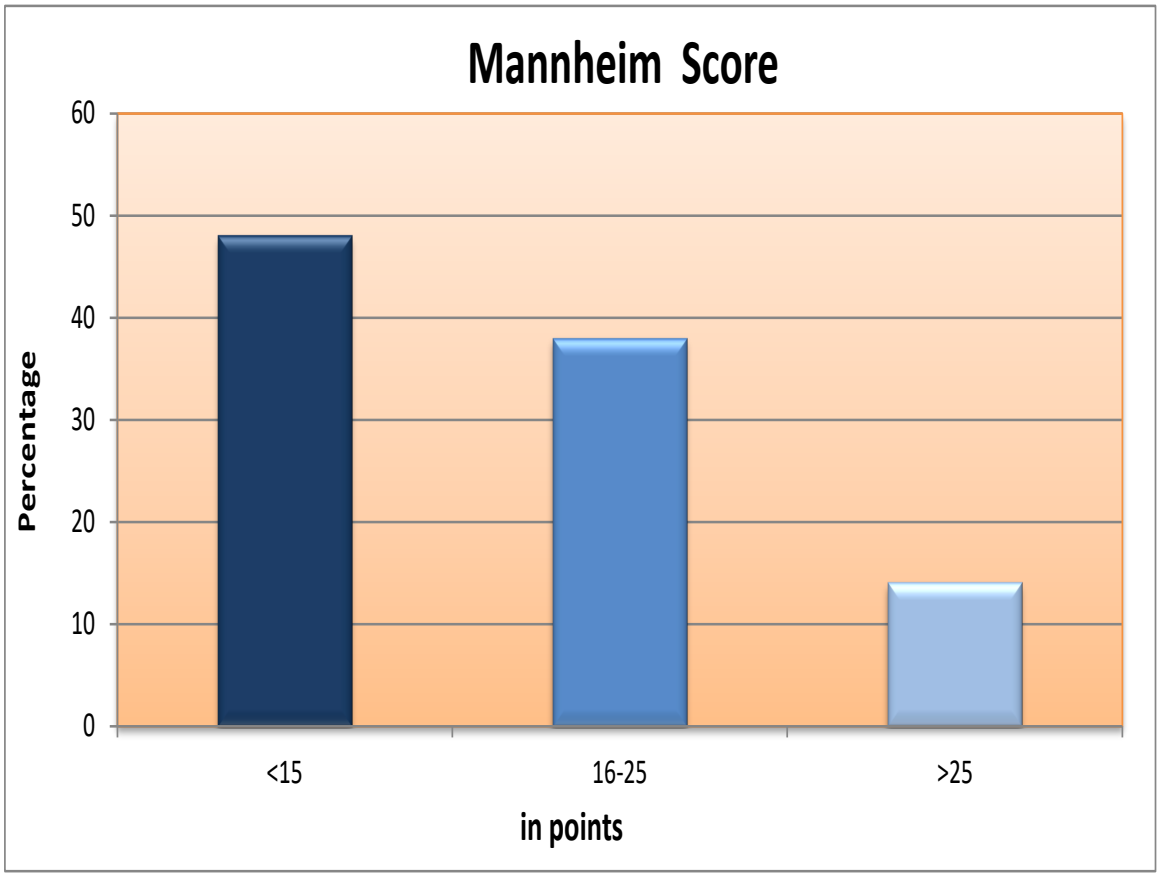
Outcome	Frequency	Percent
Died	23	15.3
Discharged	110	73.3
Wound sepsis & Discharged	17	11.3
Total	150	100



Out of 150 patients 23 (15.3%) patients were died. 17 (11.4%) were Wound sepsis and Discharged. 110 (73.3%) patients

Mannheim Score-Chart1.2

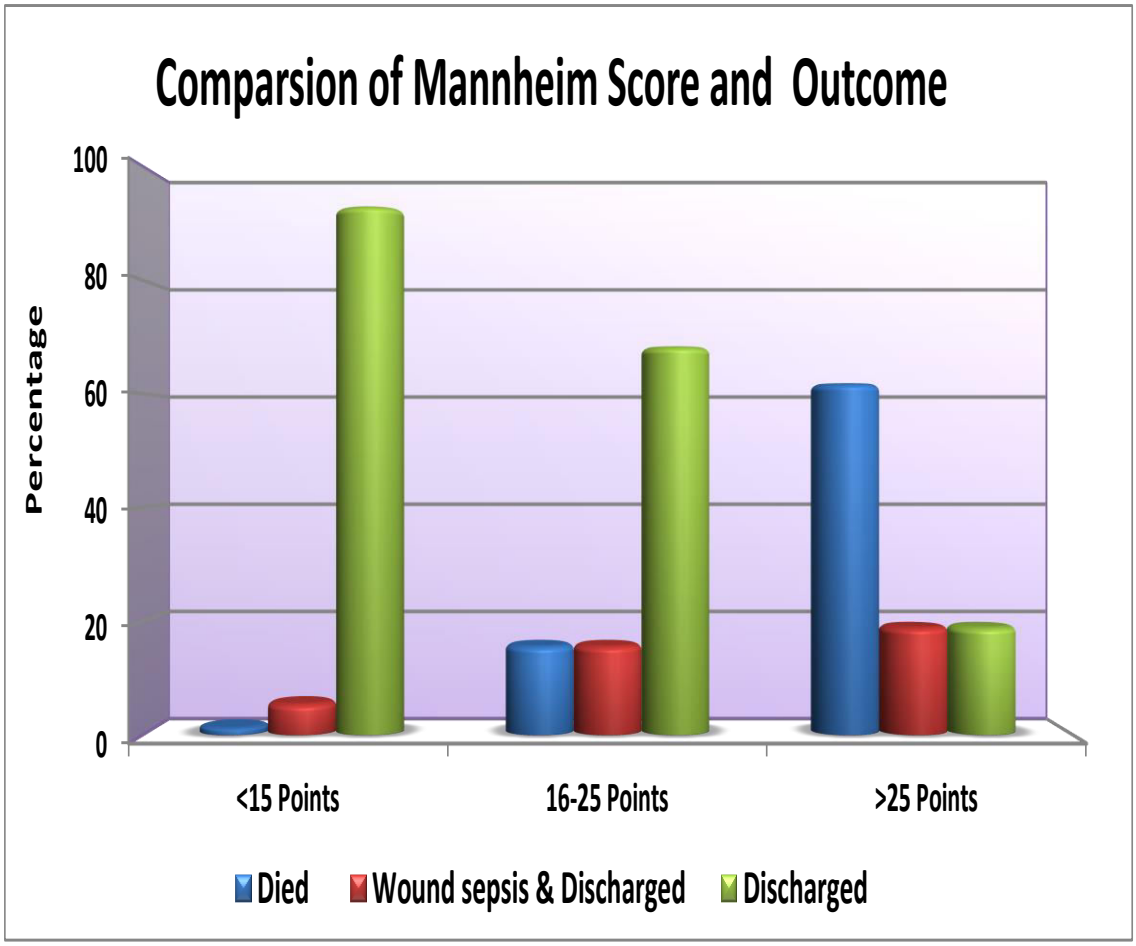
Mannheim Score	Frequency	Percent
<15	72	48.0
16-25	57	38.0
>25	21	14.0
Total	150	100



Out of 150 patients, Mannheim score for 72(48%) Patients were <15 points and 57 (28%) patients was 16-25 points. 21 (14%) patients was >25 points.

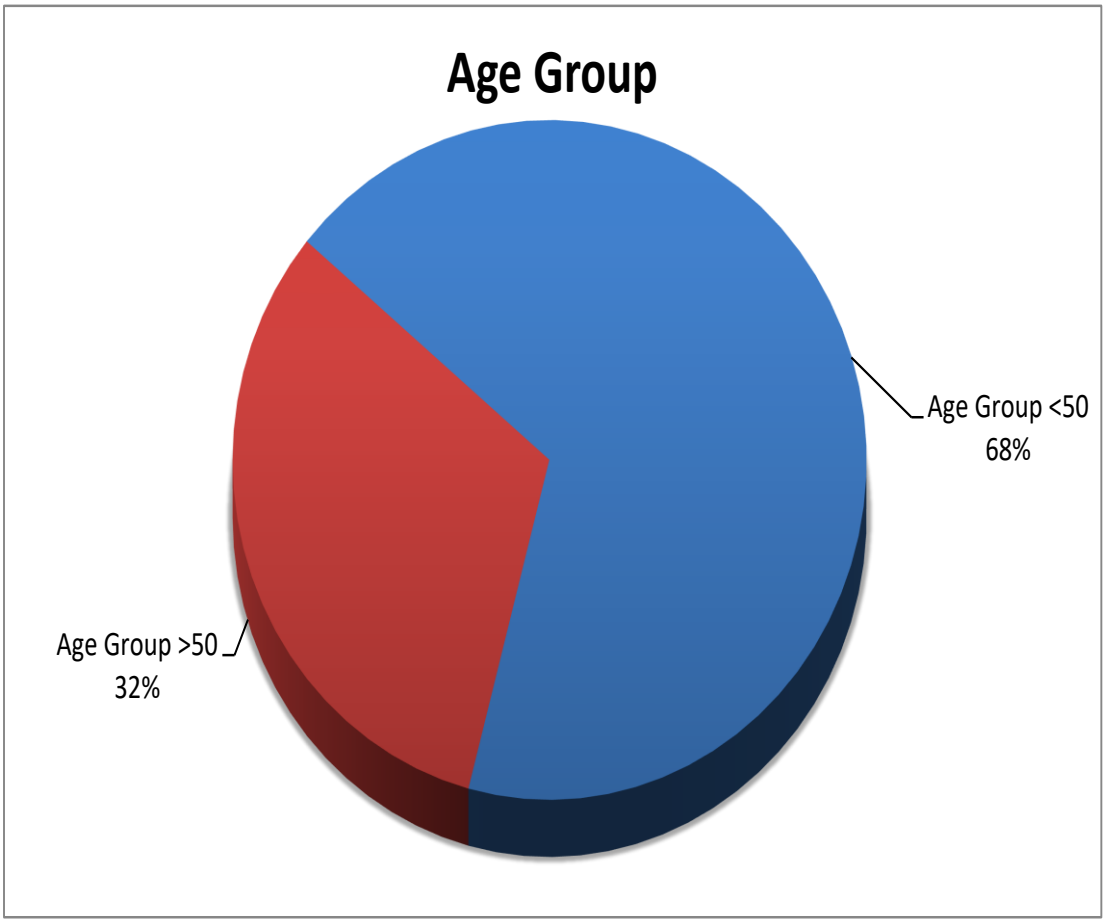
Comparison of Mannheim Score and outcome. Chart-1.3

	Mannheim Score		
	<15 Points	**16-25 Points**	**>25 Points**
Died	1	9	13
Discharged	67	39	4
Wound sepsis & Discharged	4	9	4
	72	57	21



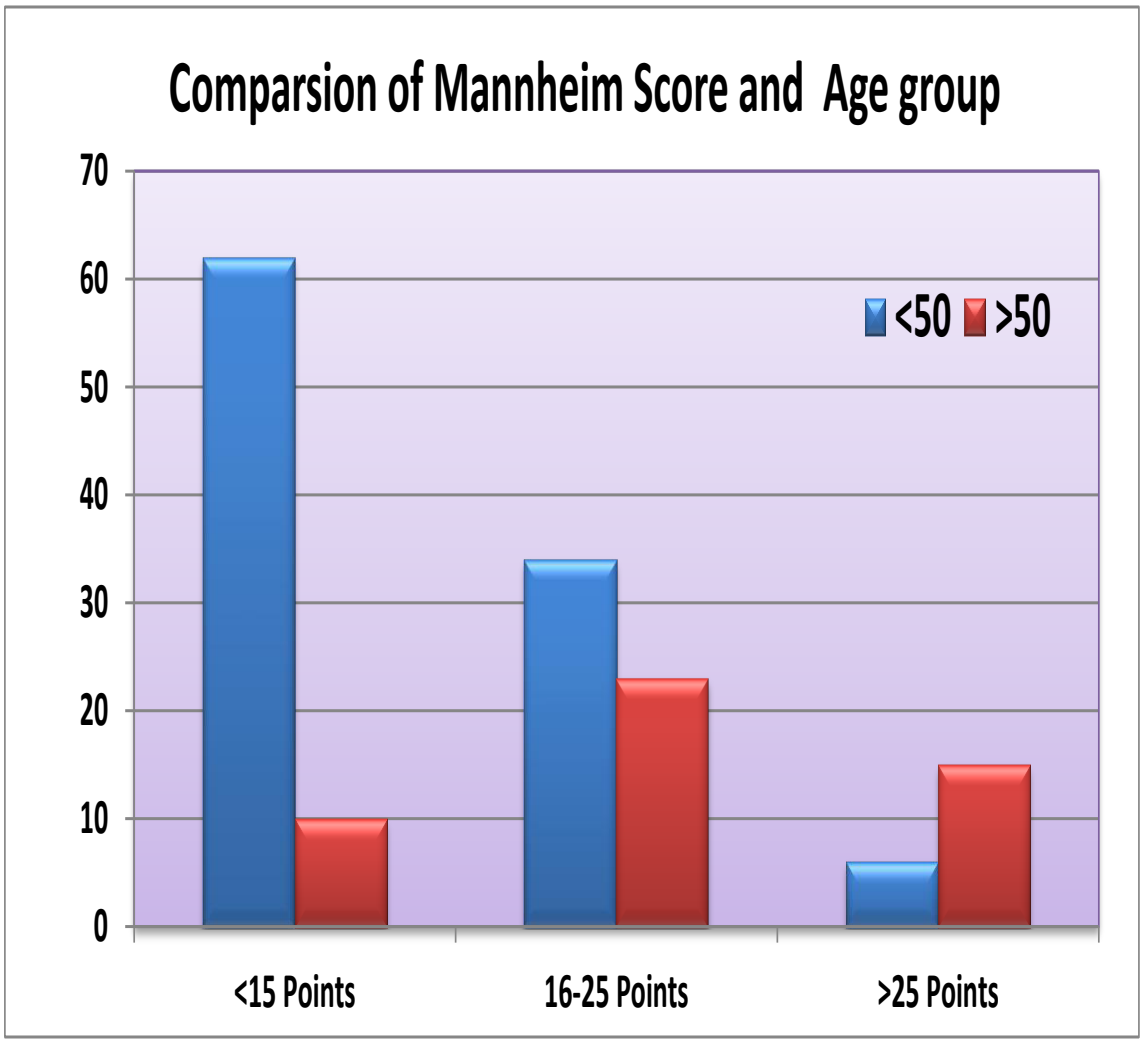
Age Group –Chart 2.1

Age Group	Frequency	Percent
<50	102	68.0
≥50	48	32.0
Total	150	100.0



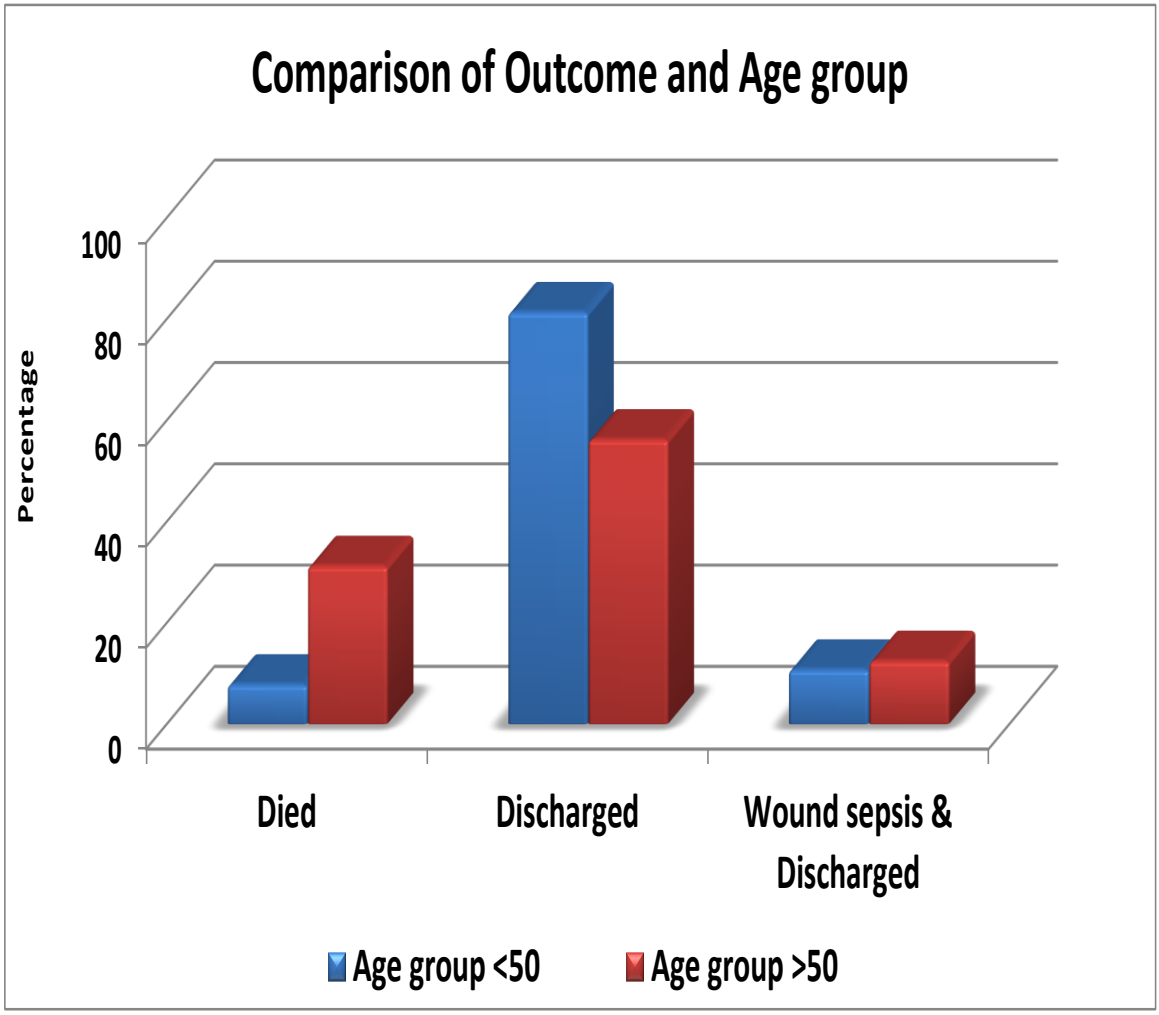
Comparison of Mannheim score and Age group - Chart-2.2

	Mannheim Score		
	<15 Points	**16-25 Points**	**>25 Points**
Age group <50	62	34	6
Age group >50	10	23	15
Total	72	57	21



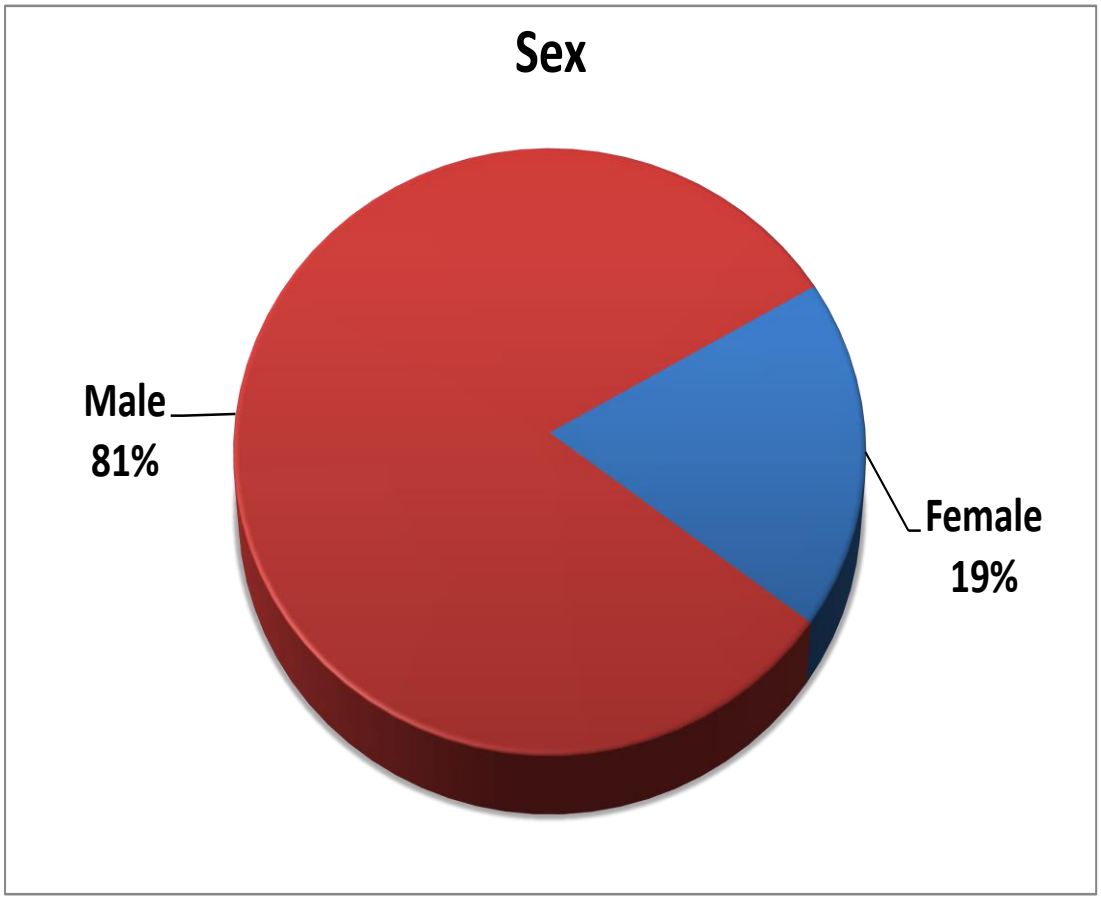
Comparison of outcome and Age group Chart -2.3

	Outcome			Total
	Died	Discharged	Wound sepsis & Discharged	
Age Group ≤50	8 (8%)	83 (81%)	11 (11%)	102
Age Group >50	15 (31%)	27 (56%)	6 (13%)	48



Sex-Chart 3.1

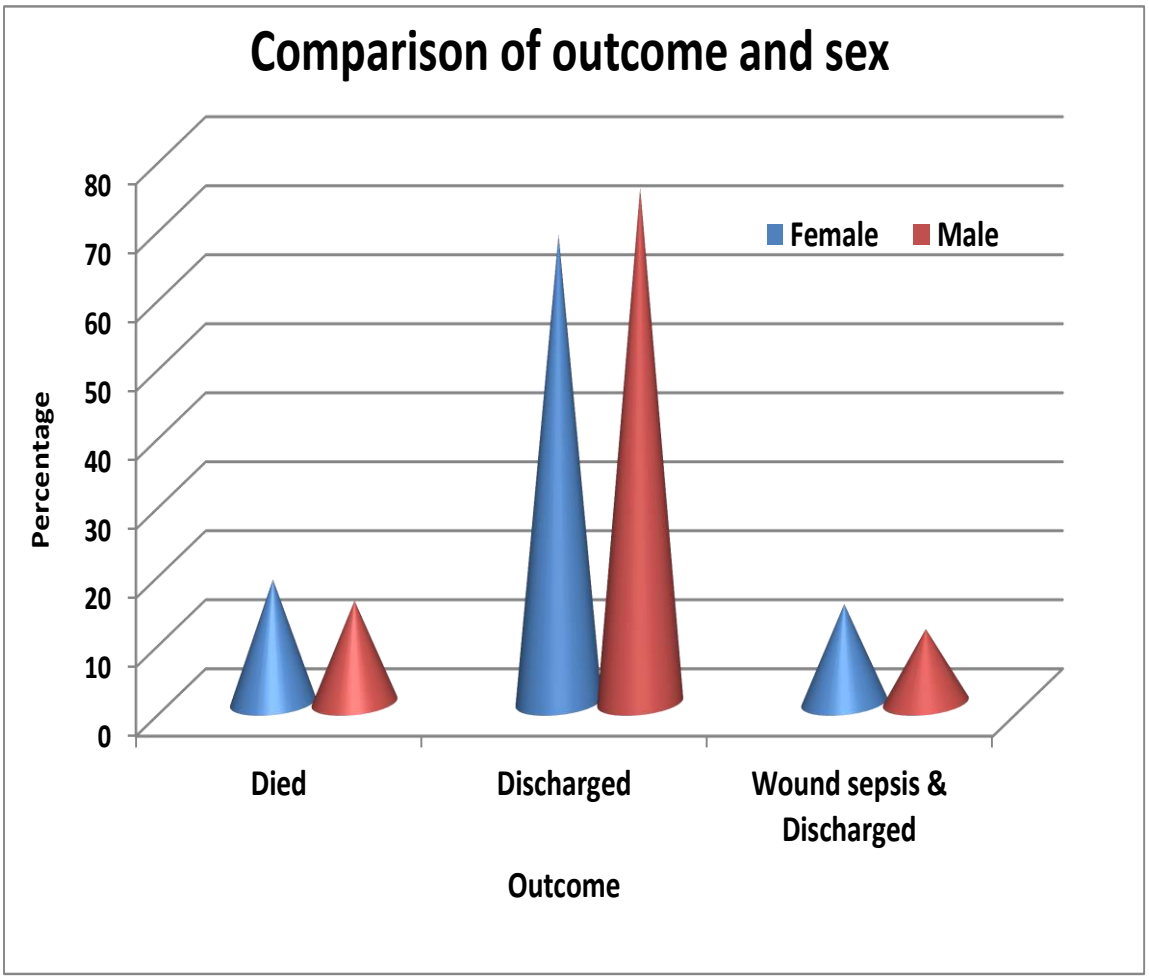
Sex	Frequency	Percent
Female	28	18.7
Male	122	81.3
Total	150	100.0



Out of 150 patients, 28 were female (18.7%) and 122 (81.3%). Group Mean age was 41.8 (±14.25) years with median of 40.00 years.

Comparison of sex and outcome Chart-3.2

	Outcome			Total
	Died	**Discharged**	**Wound sepsis & Discharged**	
Female	5 (18%)	19 (68%)	4 (14%)	28
Male	18 (15%)	91(74%)	13 (11%)	122

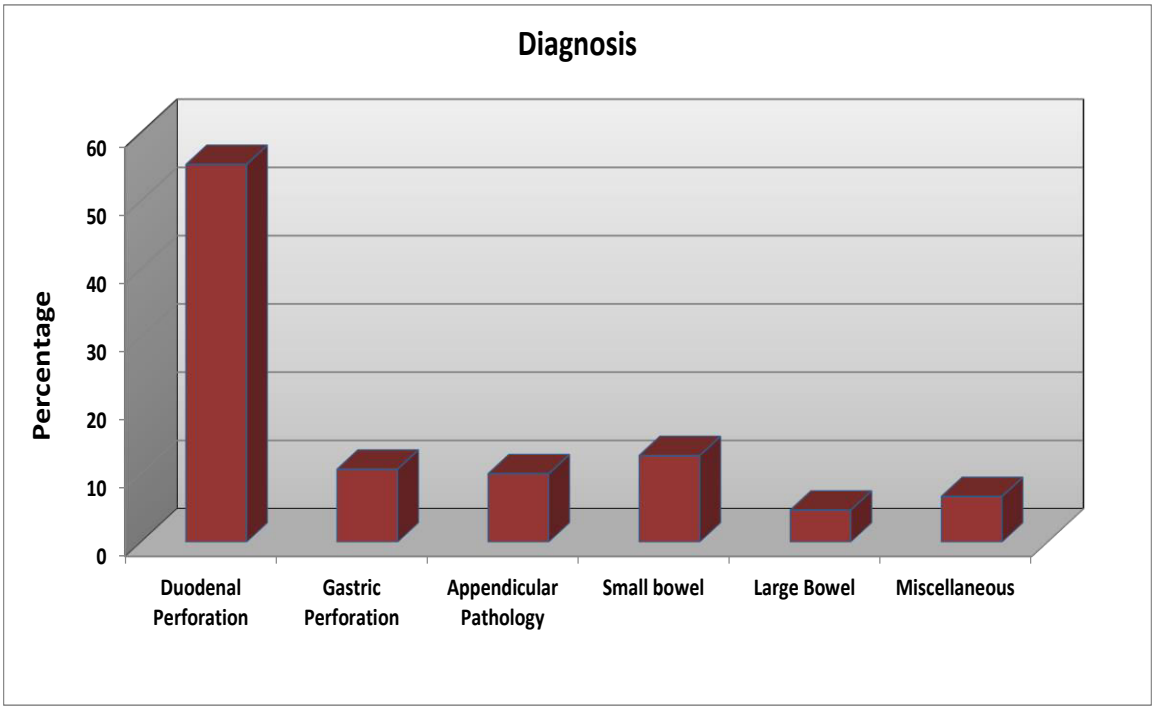


Intervention and outcome Chart-4

Day of Intervention	Outcome			Total
	Died	Discharged	Wound sepsis & Discharged	
≤ 24 hours	1 (1%)	77 (94%)	4 (5%)	82
> 24 hours	22 (32%)	33 (49%)	13 (19%)	68
	23	110	17	150

Etiological Chart 5.1

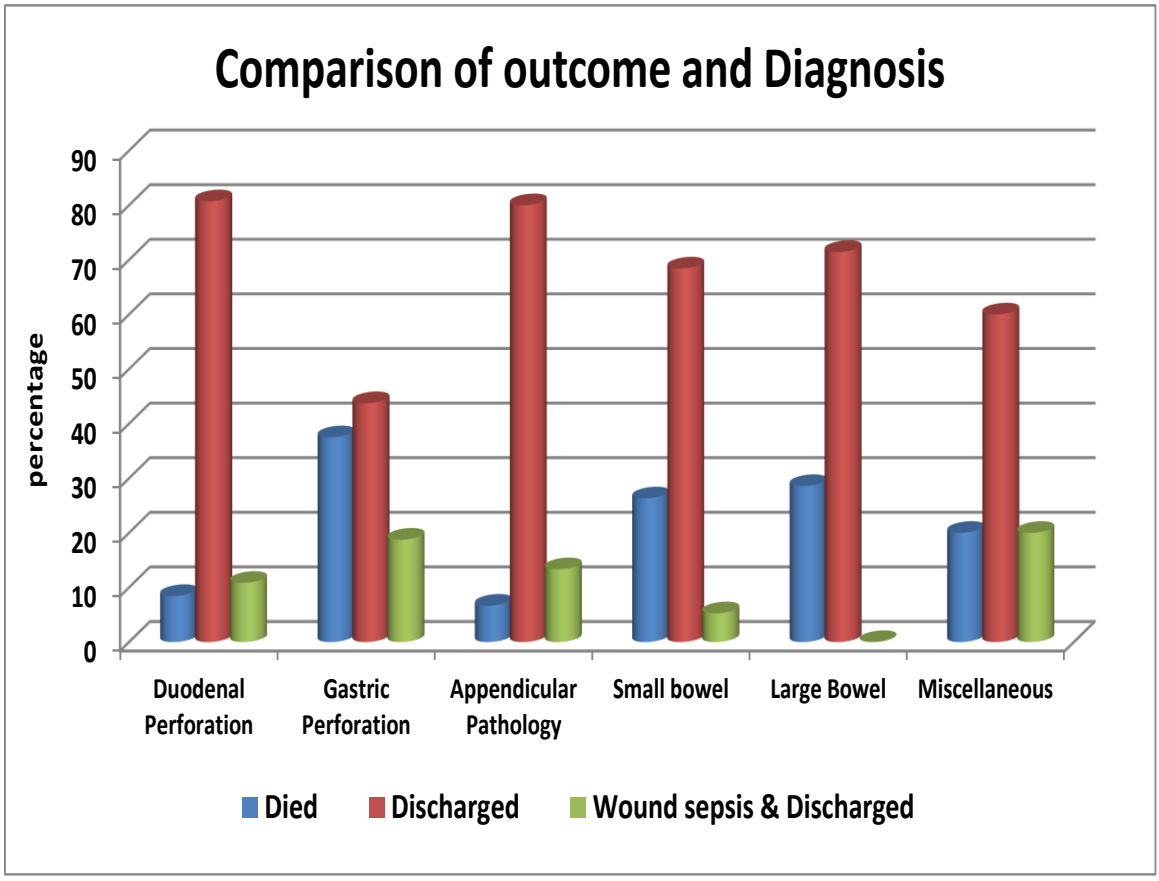
Diagnosis	Frequency	Percent
Duodenal Perforation	83	55.3
Gastric Perforation	16	10.7
Appendicular Pathology	15	10.0
Small bowel	19	12.7
Large Bowel	7	4.7
Miscellaneous	10	6.7



were evolved without any complications and were discharged from the hospital.

Comparison of outcome and Diagnosis –Chart-5.2

	Died	Discharged	Wound sepsis & Discharged	Total
Duodenal Perforation	7 (8%)	67 (81%)	9 (11%)	83
Gastric Perforation	6 (38%)	7 (44%)	3 (18%)	16
Appendicular Pathology	1 (7%)	12 (80%)	2 (13%)	15
Small bowel	5 (26%)	13 (68%)	1 (6%)	19
Large Bowel	2 (29%)	5 (71%)	0	7
Miscellaneous	2 (20%)	6 (60%)	2 (20%)	10



Comparison of risk factor of Mannheim Peritonitis index in three intervals Chart-5.3

<15 Points **48% (72/150)**						16-25 Points **38 % (57/150)**						>25 Points **14% (21/150)**					
Died (1/72) 1%		Wound sepsis & Discharged		Discharged		Died **(9/57) 16%**		Wound sepsis & Discharged		Dis charged		Died **(13/21) 76%**		Wound sepsis & Discharged		Discharged	
Patients	%		%		%		%		%		%		%		%		%
Age Group																	
Age <50	0	0	4	100	58	86.57	4	44.44	7	77.78	23	58.97	4	30.77	0	0.00	2	50.00
Age >50	1	100	0	0	9	13.43	5	55.56	2	22.22	16	41.03	9	69.23	4	100.00	2	50.00
Sex																		
Female	0	0	0	0	5	7.46	1	11.11	2	22.22	11	28.21	4	30.77	2	50.00	13	81.25
Male	1	100	4	100	62	92.54	8	88.89	7	77.78	28	71.79	9	69.23	2	50.00	3	18.75

Diagnosis																		
Non Colonic	1	100	4	100	64	96	9	100	9	100	37	95	11	85	4	100	4	100
Colonic	0	0	0	0	3	4	0	0	0	0	2	5	2	15	0	0	0	0
Day of Intervention																		
≤ 24 hours	0	0	3	75	58	86.6	1	11.1	1	11.1	17	43.6	0	0	0	0	2	50
> 24 hours	1	100	1	25	9	13.4	8	88.9	8	88.9	22	56.4	13	100.0	4	100	2	50

PROFORMA

Name	:
Age	:
Sex	:
Onset of symptoms	:
Previous Hospitalization	:
Duration of onset of symptoms to Hospital	:
D.O.A	:
D.O.D.	:
Pre OP Diagnosis	:
Post OP Diagnosis	:
Treatment	:
Date of Surgery	:
Procedure	:
Duration of Hospital stay	:

Mannheim Peritonitis Index:

	Yes	No	Score
Age > 50 Years			
Female Sex			
Organ failure			
Malignancy			
Preoperative duration of Peritonitis > 24 Hrs			
Origin of sepsis not colonic			
Diffuse generalized Peritonitis			
Exudate Clear Cloudy and purulent Fecal			
Total			
Outcome			

BIBLIOGRAPHY

1. Maddaus MA, Ahrenholz D, Simmons RL. Biología de la peritonitis. Surg Clin North Am 1988;2:471-485.

2. Wittmann DH, Walker AP, Condon RE. Peritonitis e infección intraabdominal. In: Schwartz SI, Shires GT, Spencer FC, Husser WC, Editors. Principios de cirugía. 6th ed. México: McGraw-Hill, Inc.; 1994.Pp.1493-1528.

3. Clinical files, Hospital records, *Hospital General de Durango, SSD.* Rodea-Rosas H, Athié-Gutiérrez C, Guízar-Bermúdez C, Zaldívar-Ramírez R, Flores-Martínez J. Experiencia en el tratamiento integral de 602 pacientes con sepsis abdominal. Cir Gen 1999;21:131-35.

4. Caballero RF, Acosta R, Ortíz Villalba JO. Peritonitis agudas. In: Ferreira ML, coordinator. Primeras jornadas anuales de médicos, internos, y residentes. s.l, México: Instituto de Investigación en Ciencias de la Salud;1989.Pp.567-585.

5. Pacelli F, Battista DG, Alfieri S, Piccioni E, Sgadari A, Gui D, et al. Prognosis in intra-abdominal infections. Arch Surg 1996;131(6): 641-645.

6. Barriere SL, Lowry SF. An overview of mortality risk prediction in sepsis. Crit Care Med 1995;23(2):376-393.

7. Koperna T, Schulz F. Prognosis and treatment of peritonitis. Do we need new scoring systems? Arch Surg 1996;131:180-186.

8. Knaus WA, Wagner DP, Draper EA, Zimmerman JE, Berger M, Bastos PG, et al. The APACHE III prognostic system. Risk prediction of hospital mortality for critically ill hospitalized adults. Chest 1991;100(6):1619-1636.

9. Sawyer RG, Rosenlof LK, Adams RB, May AK, Spengler MD, Pruett TL. Peritonitis into the 1990s: changing pathogens and changing strategies in the critically ill. Am Surg 1992 Feb;58(2):82-87.

10. Lemeshow S, Teres D, Spitz Avrunin J, Pastides H. A comparison of methods to predict mortality of intensive care unit patients. Crit Care Med 1987;15(8):715-722.

11. Velásquez-Díaz G, coordinator. Lineamientos y criterios de estructura progresiva para la integración de recursos humanos en hospitales generales de 30 a 180 camas censables. s.l.: Dirección de Desarrollo de los Servicios de Salud. Dirección General de Regulación de los Servicios de Salud. Subsecretaría de Regulación y Fomento Sanitario. México: Secretaría de Salud;2000:Pp.77-83.

12. Dirección General de Estadística e Informática. Directorio de Unidades 1999. México, D.F.: Secretaría de Salud; 2000.

13. Linder MM, Wacha H, Feldmann U, Wesch G. Streifensand RA, Gundlach E. Der Mannheimer peritonitis-index. Ein instrument zur intraoperativen prognose der peritonitis. Chirurg 1987;58(2):84–92.

14. Billing A, Fröhlich D, Schildberg FW. Prediction of outcome using the Mannheim peritonitis index in 2003 patients. Br J Surg 1994;81: 209-213.

15. Zeitoun G, Laurent A, Rouffet F, Hay J, Fingerhut A, Paquet J, Peillon C, Research TF. Randomized clinical trial: multicentre, randomized clinical trial of primary *versus* secondary sigmoid resection in generalized peritonitis complicating sigmoid diverticulitis. Br J Surg 2000 Oct;87(10):1366-1374.

16. Hut'an M, Poticny V, Balaz P. Modern trends in the treatment of diffuse peritonitis. Rozhl Chir 2000 Apr;79(4):171-174.

17. Seiler CA, Brugger L, Forssmann U, Baer HU, Buchler MW. Conservative surgical treatment of diffuse peritonitis. Surgery 2000 Feb;127(2):178-184.

18. Yilmazlar T, Toker S, Zorluoglu A. Non-traumatic colorectal perforations. Int Surg 1999 Apr-Jun;84(2):155-158

19. Bergamaschi R, Marvik R, Johnsen G, Thoresen JE, Ystgaard B, Myrvold HE. Open *vs* laparoscopic repair of perforated peptic ulcer. Surg Endosc 1999 Jul;13(7):679-682.

20. Brugger LE, Seiler CA, Mittler M, Balsiger B, Feodorovici M, Baer HU, Buchler MW. New approaches to the surgical treatment of diffuse peritonitis. Zentralbl Chir 1999;124(3):181-186.

21. Seiler CA, Brugger L, Maurer CA, Renzulli P, Buchler MW. Peritonitis in diverticulitis: the Bern concept. Zentralbl Chir 1998;123(12): 1394-1394.

22. Liverani A, Correnti SF, Paganelli MT, Antonini G, Mercati U. Mannhein index in the prognosis and treatment of acute peritonitis. Minerva Chir 1998 May;53(5):385-389.

23. Moesgaard F, Jensen LS, Christiansen PM, Thorlacius-Ussing O, Nielsen KT, Rasmussen NR, Bardram L, Nielsen HJ. The effect of ranitidine on postoperative infectious complications following emergency colorectal surgery: a randomized, placebo-controlled, doubleblind trial. Inflamm Res 1998 Jan;47(1):12-17.

24. Bosscha K, Reijnders K, Hulstaert PF, Algra A, van der Werken C. Prognostic scoring systems to predict outcome in peritonitis and intra- abdominal sepsis. Br J Surg 1997 Nov;84(11):1532-1534.

25. Ohmann C, Hau T. Prognostic indices in peritonitis. Hepatogastroenterology 1997 Jul-Aug;44(16):937-946.

26. Grunau G, Heemken R, Hau T. Predictors of outcome in patients with postoperative intra-abdominal infection. Eur J Surg 1996 Aug;162(8):619-625.

27. Gotzinger P, Gebhard B, Wamser P, Sautner T, Huemer G, Fugger R. Revision of diffuse peritonitis: planned *versus* on demand. Langenbecks Arch Chir 1996;381(6):343-347.

28. Ermolov AS, Bagdat'ev VE, Chudotvortseva EV, Rozhnov AV. Evaluation of the Mannheim peritonitis index. Vestn Khir Im I I Grek 1996;155(3):22-23.

29. Burger JA, Schoffel U, Sach M, Jacobs E, Kownatzki E, von Specht BU, Farthmann EH. Effects of peritonitis exudates on chemotaxis and phagocytosis of human neutrophils. Eur J Surg 1995 Sep;161(9):647-653.

30. Schoeffel U, Jacobs E, Ruf G, Mierswa F, von Specht BU, Farthmann EH. Intraperitoneal micro-organisms and the severity of peritonitis. Eur J Surg 1995 Jul;161(7):501-508.

31. Demmel N, Maag K, Osterholzer G. The value of clinical parameters for determining the prognosis of peritonitis-validation of the Mannheim Peritonitis Index. Langenbecks Arch Chir 1994;379(3):152-158.

32. Demmel N, Muth G, Maag K, Osterholzer G. Prognostic scores in peritonitis: the Mannheim Peritonitis Index or APACHE II? Langenbecks Arch Chir 1994;379(6):347-352.

33. Nespoli A, Ravizzini C, Trivella M, Segala M. The choice of surgical procedure for peritonitis due to colonic perforation. Arch Surg 1993 Jul;128(7):814-818.

34. Ohmann C, Wittmann DH, Wacha H. Prospective evaluation of prognostic scoring systems in peritonitis. Peritonitis Study Group. Eur J Surg 1993 May;159(5):267-74.

35. Altaca G, Sayek I, Onat D, Cakmakci M, Kamiloglu S. Risk factors in perforated peptic ulcer disease: comparison of a new score system with the Mannheim Peritonitis Index. Eur J Surg 1992 Apr;158(4): 217-221.

36. Rogy M, Fugger R, Schemper M, Koss G, Schulz F. The value of 2 distinct prognosis scores in patients with peritonitis. The Mannheim peritonitis index *versus* the Apache II score. Chirurg 1990 Apr;61(4):297-300.

37. Krenzien J, Lorenz W. Scoring systems for severe intra-abdominal infections. Zentralbl Chir 1990;115(17):1065-1079.

38. Eypasch E, Spangenberger W, Bouillon B, Moser KH, Vestweber KH, Troidl H. Score systems, their importance for the intensive care patient. Langenbecks Arch Chir Suppl II Verh Dtsch Ges Chir 1989;813-817.

39. Fugger R, Rogy M, Herbst F, Schemper M, Schulz F. Validation study of the Mannheim Peritonitis Index. Chirurg 1988 Sep;59(9):598-601.

40. Schirrmacher E, Seifert J. The Mannheim peritonitis index. Its reliability for the assessment of prognosis in peritonitis patients. Fortschr Med 1988 Jul 30;106(22):454-456.

41. Sautner T, Götzinger, Redl-Wenzl EM, Dittrich K, Felfernig M, Sporn P, et al. Does reoperation for abdominal sepsis enhance the inflammatory host response? Arch Surg 1997;132:250-255.

42. Navez B, Tassetti V, Scohy JJ, Mutter D, Guiot P, Evrard S, et al. Laparoscopic management of acute peritonitis. Br J Surg 1997;85: 32-36.

43. Rodloff AC, Kujath P, Lunstedt B, Gaus W. Vergleichende untersuchung zum kosten-/effektivitatsverhaltnis einer initialen therapie mit imipenem/cilastin bei der sekundaren peritonitis. Chirurg 1998;69(10): 1093-1100.

44. Kriwanek S, Gschwntler M, Beckerhinn P, Armbruster C, Roka R. Langzeitergebnisse nach Hartmann-operationen. Chirurg 1999;70(1): 49-53.

45. Bruch HP, Woltmann A, Eckmann C. Chirurgisches management bei peritonitis und sepsis. Zentralbl Chir 1999;124(3):176-180.

46. Rodríguez-Gojón GH, Pérez-García R, Poblano-Morales M, Rodríguez- Brambilia V, Chavero-Garcia V. Factores pronósticos asociados a mortalidad en pacientes con sepsis abdominal tratados en la unidad de terapia intensiva. Cir Cir 1999;67(6):205-207.

47. Demmel N, Osterholzer G, Gunther B. Differentiated treatment strategy for peritonitis: single stage closure with drainage or open with programmed reinterventrion/lavage? Zentralbl Chir 1993;1187(7): 395-400.

48. Kriwanek S, Armbruster C, Beckerhinn P, Dittrich K. Prognostic factors for survival in colonic perforation. Int J Colorectal Dis 1994;9(3):158-162.

49. Winkeltau GJ, Bertram P, Braun J, Schumpelick V. Differentiated surgical therapy of diffuse peritonitis–a prospective study. Leber Magen Darm 1996;26(3):144-148.

50. Winkeltau GJ, Tons C, Bertram P, Schumpelick V. Stage-oriented antibiotic therapy of peritonitis. Langenbecks Arch Chir 1997;382(4 Suppl 1):S22-25.

51. Tassetti V, Valvano L, Navez B, Mutter D, Scohy JJ, Evrard S, Marescaux J. Perforated peptic ulcer and laparoscopic treatment. Minerva Chir 1998;53(10):777-780.

52. Yilmazlar T, Toker S, Zorluo glu A. Non traumatic colorectal perforations. Int Surg 1999;84(2):155-158.

53. Van Laarhoven CJ, Hoofwijk AG, van Vroonhoven TJ. The Mannheim peritonitis index: a valuable method in predicting the outcome of severe peritonitis? Neth J Surg 1988;40(6):168.

54. Copes WS, Champion HR, Sacco WJ, Lawnick MM, Keast SL, Bain LW. The injury severity score revisited. J Trauma 1988;28(1):69-77.

55. Farthmann EH, Schoffel U. Epidemiology and pathophysiology of intraabdominal infections (IAI). Infection 1998;26(5):329-334.

56. Bailey & love'short practice of surgery, 25th edition,

57. Maingot's abdominal Operation, 11th ediction

58. Shackelford's surgery of alimentary tract, 6th edition

59. Mastery of Surgery, 5th Edition, Volume-1, p.27-29.

60. Sabiston's text book of surgery, 18th Edition

61. Schwart'zs principles of surgery, 8th edition

62. Diagnostic Radiology by Grainger and allison,5th edition

63. Essentil surgical practice by Sir Alfred cuschieri, 4th Edition

64. Current surgical diagnosis and treatment, 11th edition

65. Imaging of Acute Abdomen Radiologic clinics of North America

66. Japanes journal of gastroenterological surgery, Vol.37, No.1, p.7-13. (2004)

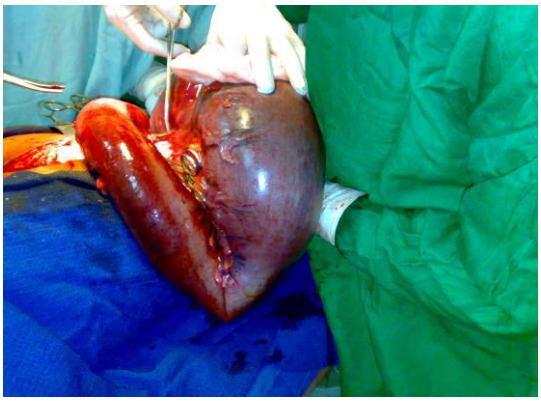
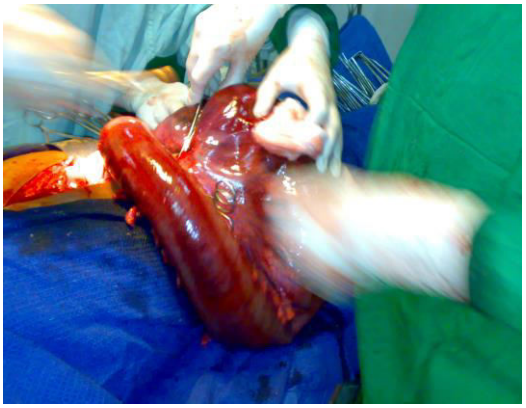
67. Ebonyi Medical Journal, vol.6, (i) 2006, p.3-8.

68. AJS, Volume-81, issue-2

PEROPERATIVE PICTURES





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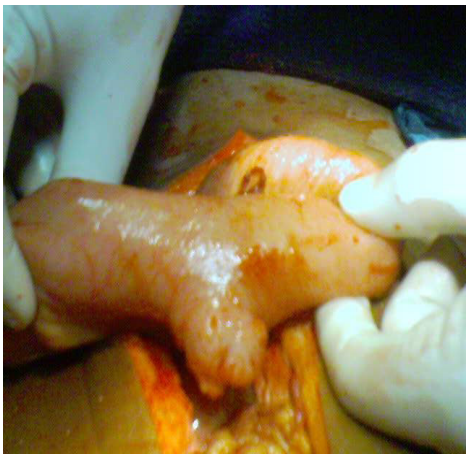
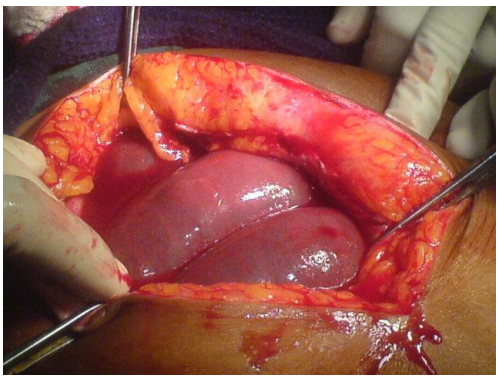
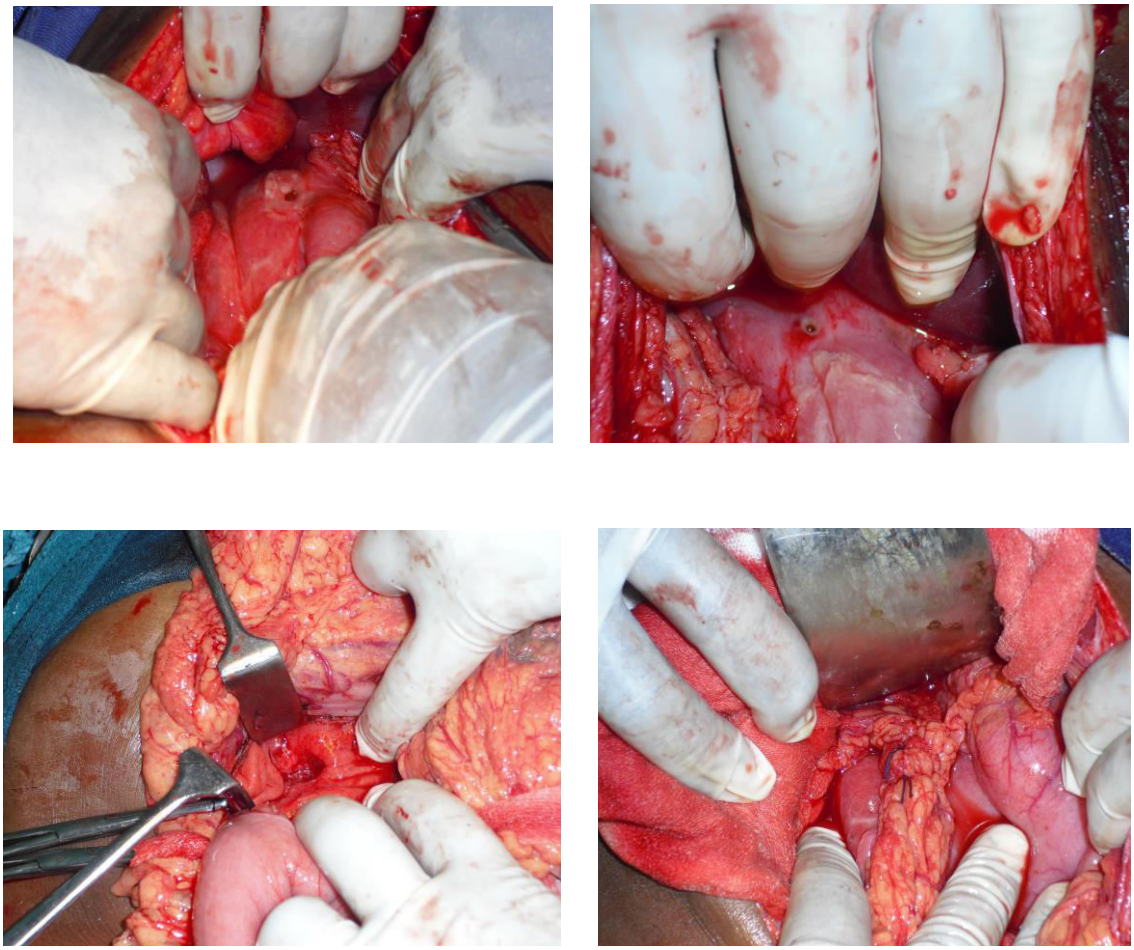


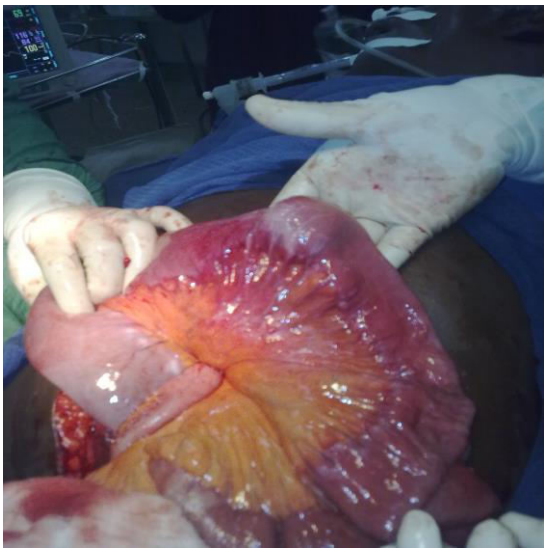
PICTURES SHOWING: MECKEL DIVERTICULUM

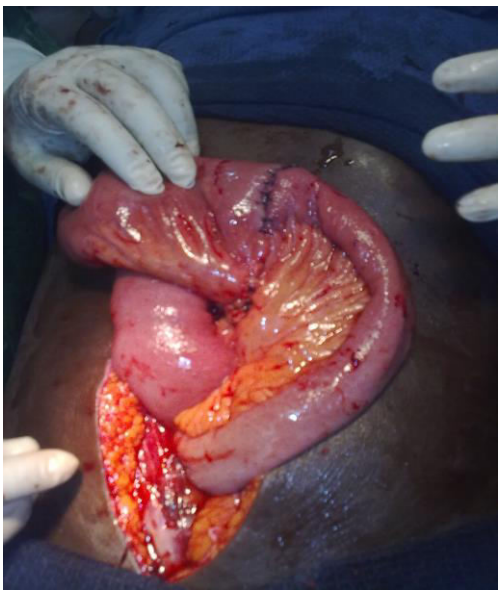



INTESTINAL OBSTRUCTION: RESECTION ANASTOMOSIS



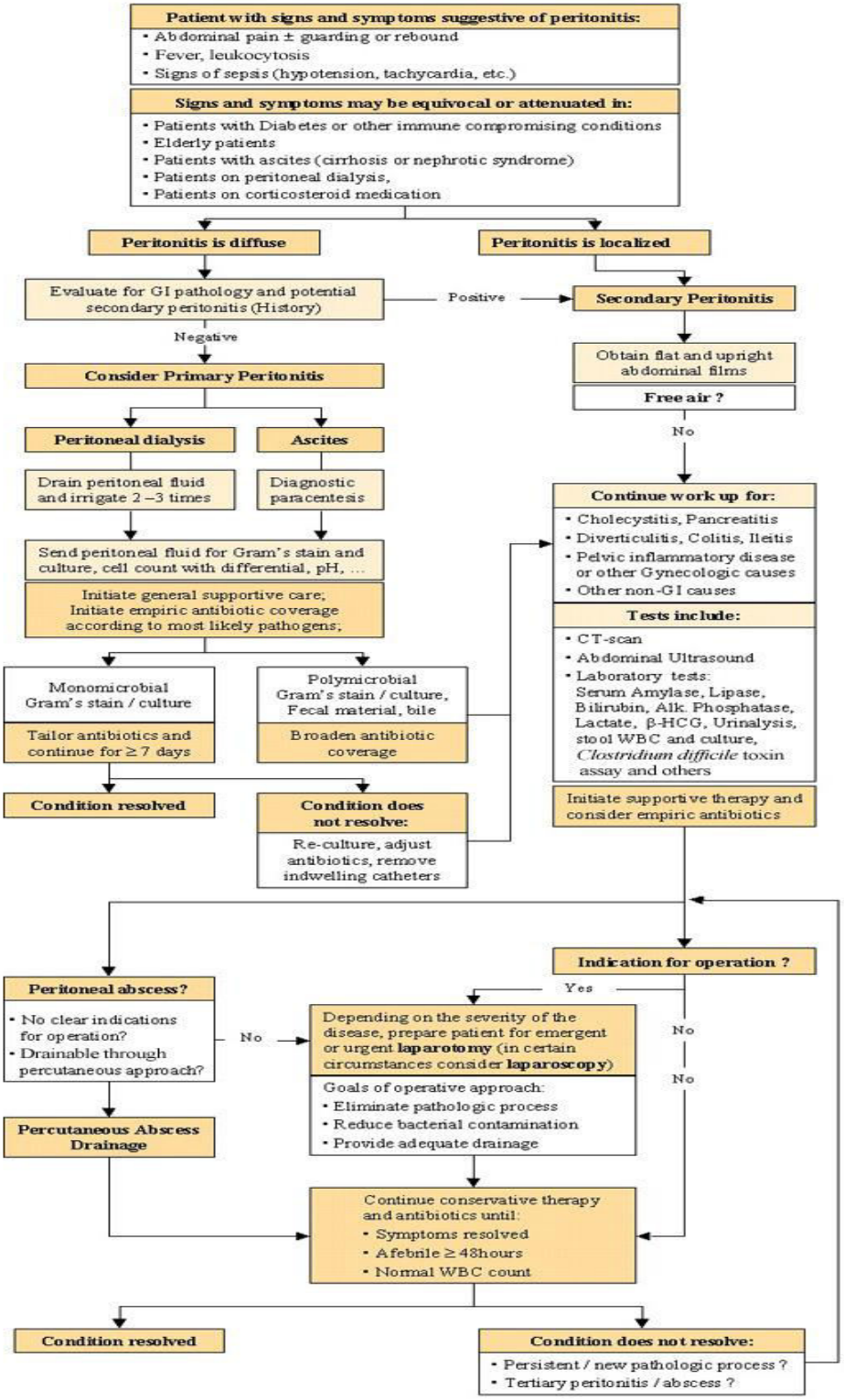
Duodenal and Gastric perforation and perforation closure.




Intussusception -Resection Anastomosis caused by Fibroma

Diagnostic and therapeutic approach to peritonitis and peritoneal abscess:



MASTER CHART

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
1	Mr.Kumar	28	M	1	9	10	Duodenal perforation	Laparotomy perforation closure	Discharged
2	Mr.Pradeep	20	M	1	9	10	Duodenal perforation	Laparotomy perforation closure	Discharged
3	Mr.Chinnappa	50	M	2	31	20	Duodenal perforation	Laparotomy perforation closure	Discharged
4	Mr.Duraiswamy	45	M	1	9	20	Prepyloric perforation	Laparotomy perforation closure	Wound sepsis & Discharged
5	Mr.Devaraj	65	M	1	12	15	Duodenal perforation	Laparotomy perforation closure	Discharged
6	Mr.Kuppan	45	M	3		27	Ca.Rectum	End colostomy	Died
7	Mr.Sengeni	45	M	1	10	10	Ruptured Liver abscess	Peritoneal lavage	Discharged
8	Mr.Raja	20	M	3	25	16	Gangrenous appendicitis	Peritoneal lavage	Discharged
9	Mr.Rathinam	65	M	2	25	29	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
10	Mr.Durai	60	M	1	11	15	Gasrtic perforation	Laparotomy perforation closure	Discharged
11	Mr.Prasanth	15	M	1	13	10	Duodenal perforation	Laparotomy perforation closure	Discharged
12	Mrs.Malliga	55	F	1	16	18	Duodenal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
13	Mr.Gangadharan	58	M	2	13	19	Duodenal perforation	Laparotomy perforation closure	Discharged
14	Mr.Saranraj	14	M	1	9	10	Duodenal perforation	Laparotomy perforation closure	Discharged
15	Mr.Shanmugam	48	M	2	15	14	Duodenal perforation	Laparotomy perforation closure	Discharged
16	Mr.Ramakrishnan	52	M	1	9	15	Duodenal perforation	Laparotomy perforation closure	Discharged
17	Mr.Dhanamal	70	F	2	31	30	Prepyloric perforation	Laparotomy perforation closure	Wound sepsis & Discharged
18	Mr.Settu	40	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
19	Mr.Gunapatham	35	M	2	10	20	Appendicular perforation	Peritoneal lavage & appendicectomy	Discharged
20	Mr.Kuttiammal	35	F	3	13	26	Ileal gangrene	Resection anastomosis	Discharged
21	Mr.Perumal	47	M	1	9	10	Duodenal perforation	Laparotomy perforation closure	Discharged
22	Mr.Badrulan	45	M	1	13	14	Duodenal perforation	Laparotomy perforation closure	Discharged
23	Mr.Arumugam	35	M	3	11	20	Appendicular perforation	Peritoneal lavage appendicectomy	Discharged
24	Mr.Kathirvel	35	M	4	13	14	Ileal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
25	Mrs.Kuppammal	40	F	7	11	25	Ileal perforation	Laparotomy perforation closure	Discharged
26	Mr.Ravi	32	M	1	10	14	Duodenal perforation	Laparotomy perforation closure	Discharged
27	Mrs.Chokkammal	63	F	7	16	30	Ileal perforation	Laparotomy perforation closure	Discharged
28	Mr.Subramani	40	M	3	12	20	Gangrenous appendicitis	Peritoneal lavage & appendicectomy	Discharged
29	Mr.Manivannan	29	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
30	Mr.Elumalai	52	M	1	10	15	Duodenal perforation	Laparotomy perforation closure	Discharged
31	Mr.Mohanraj	25	M	3	15	20	Appendicular perforation	Peritoneal lavage & appendicectomy	Wound sepsis & Discharged
32	Mr.Veerappan	40	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
33	Mr.Rajesh	17	M	1	9	10	Duodenal perforation	Laparotomy perforation closure	Discharged
34	Mr.Karthikeyan	40	M	2	15	16	Appendicular perforation	Peritoneal lavage & appendicectomy	Discharged
35	Mr.Sridhar	55	M	2	12	19	Duodenal perforation	Laparotomy perforation closure	Discharged
36	Mr.Muniyan	32	M	1	8	16	Duodenal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
37	Mr.Mani	49	M	1	10	8	Duodenal perforation	Laparotomy perforation closure	Discharged
38	Mrs.Latha	48	F	1	9	15	Duodenal perforation	Laparotomy perforation closure	Discharged
39	Mr.Vaithi	60	M	1	10	9	Duodenal perforation	Laparotomy perforation closure	Discharged
40	Mr.Valliammal	60	F	3		30	Gasrtic perforation	Laparotomy perforation closure	Died
41	Mr.Arul Balan	43	M	5	20	20	Ruptured Liver abscess	Peritoneal lavage	Discharged
42	Mr.Elumalai	50	M	3		32	Ileal gangrene	Ileostomy	Died
43	Mr.Vasu	64	M	4		25	Prepyloric perforation	Laparotomy perforation closure	Died
44	Mr.Baskaran	35	M	3	10	14	Appendicular perforation	Peritoneal lavage & appendicectomy	Discharged
45	Mr.Valliammal	60	F	2	10	24	Ileal perforation	Laparotomy perforation closure	Discharged
46	Mrs.Rani	35	F	1	14	15	Duodenal perforation	Laparotomy perforation closure	Discharged
47	Mr.Ponnurangam	62	M	3		25	Duodenal perforation	Laparotomy perforation closure	Died
48	Mr.Loganathan	40	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
49	Mr.Ramamoorthy	35	M	2	28	10	Sigmoid volvulus	Laparotomy resection colostomy	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
50	Mr.Ravi	40	M	2	19	10	Sigmoid volvulus	Laparotomy resection colostomy	Discharged
51	Mr.Balaji	30	M	1	10	14	Duodenal perforation	Laparotomy perforation closure	Discharged
52	Mr.Ravi	20	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
53	Mr.Parasuramman	42	M	1	14	14	Duodenal perforation	Laparotomy perforation closure	Discharged
54	Mr.Karthik	18	M	1	5	16	Appendicular perforation	Peritoneal lavage & appendicectomy	Discharged
55	Mr.Ellappan	40	M	3		20	Prepyloric perforation	Laparotomy perforation closure	Died
56	Mr.Perumal	22	M	1	11	10	Duodenal perforation	Laparotomy perforation closure	Discharged
57	Mr.Kulluchami	59	M	1	37	13	RTA blunt injury abdomen pelvic fracture	Laparotomy bladder injury repair	Discharged
58	Mr.Subrayan	25	M	1	12	15	Duodenal perforation	Laparotomy perforation closure	Discharged
59	Mr.Dharman	32	M	3	10	20	Duodenal perforation	Laparotomy perforation closure	Discharged
60	Mr.Sugumar	25	M	1	10	14	Duodenal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
61	Mr.Mani	42	M	1	8	10	Duodenal perforation	Laparotomy perforation closure	Discharged
62	Mr.Umapathy	60	M	3	12	25	Ruptured Liver abscess	Peritoneal lavage	Discharged
63	Mr.Hari	34	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
64	Mr.Muthuvel	35	M	2	17	19	Antral perforation	Laparotomy perforation closure	Discharged
65	Mr.Durai	40	M	1	12	14	Duodenal perforation	Laparotomy perforation closure	Discharged
66	Mr.Selvam	40	M	1	13	10	Duodenal perforation	Laparotomy perforation closure	Discharged
67	Mr.Elumalai	16	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
68	Mr.Senu	35	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
69	Mr.Shaikali	45	M	7	24	20	Ruptured Liver abscess	Peritoneal lavage	Wound sepsis & Discharged
70	Mrs.Amirthammal	45	F	3	14	19	Sigmoid volvulus	Resection anastomosis	Discharged
71	Mr.Moorthy	42	M	1	20	10	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
72	Mrs.Venda	42	F	1	12	9	Duodenal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
73	Mr.Munuswamy	56	M	3	15	25	Prepyloric perforation	Laparotomy perforation closure	Wound sepsis & Discharged
74	Mr.Bakiya	45	F	2	15	25	Duodenal perforation	Laparotomy perforation closure	Discharged
75	Mr.Arumugam	45	M	5	26	20	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
76	Mr.Krishnan	19	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
77	Mr.Devaraj	44	M	1	15	16	Prepyloric perforation	Laparotomy perforation closure	Discharged
78	Mr.Ramesh	33	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
79	Mr.Munuswamy	65	M	5	15	25	Duodenal perforation	Laparotomy perforation closure	Discharged
80	Mr.Devaraj	30	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
81	Mr.Ramadass	50	M	2	13	14	Duodenal perforation	Laparotomy perforation closure	Discharged
82	Mrs.Alamelu	60	F	7		30	Ruptured Liver abscess	Peritoneal lavage	Died
83	Mr.Arumugam	30	M	2	17	20	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
84	Mr.Kanniappan	47	M	1	12	10	Duodenal perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
85	Mr.Logannathan	40	M	2	15	20	Duodenal perforation	Laparotomy perforation closure	Discharged
86	Mr.Gurunathan	50	M	2	11	19	Prepyloric perforation	Laparotomy perforation closure	Discharged
87	Mr.Narayanan	40	M	1	12	14	Duodenal perforation	Laparotomy perforation closure	Discharged
88	Mr.Chandran	35	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
89	Mrs.Devi	28	F	1	12	9	Duodenal perforation	Laparotomy perforation closure	Discharged
90	Mr.Venugopal	65	M	1	13	19	Duodenal perforation	Laparotomy perforation closure	Discharged
91	Mr.Vasu	60	M	4	13	21	Ileal gangrene	Resection anastomosis	Discharged
92	Mr.Elumalai	60	M	1	14	25	Ileal gangrene	Resection anastomosis	Discharged
93	Mrs.Valliammal	78	F	7	21	30	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
94	Mr.Balu	50	M	1	10	19	Duodenal perforation	Laparotomy perforation closure	Discharged
95	Mr.Ravichandran	70	M	3		25	Duodenal perforation	Laparotomy perforation closure	Died
96	Mr.Pandiyan	52	M	1	11	19	Duodenal perforation	Laparotomy perforation closure	Discharged
97	Mrs.Meenatchi	57	F	1	14	21	Ileal gangrene	Resection	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
								anastomosis	
98	Mr.Srinivasan	50	M	2		25	Gasrtic perforation	Laparotomy perforation closure	Died
99	Mr.Perumal	53	M	5		31	Ileal perforation	Laparotomy perforation closure	Died
100	Mrs.Lalitha	45	F	1	13	26	Small bowel adhesion & banding	Laparotomy resection anastomosis	Discharged
101	Mr.Munuswamy	65	M	3		30	Gasrtic perforation	Laparotomy perforation closure	Died
102	Mr.Raja	24	M	1	11	14	Appendicular perforation	Laparotomy & Appendicectomy	Discharged
103	Mr.Pattabi	40	M	3	18	20	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
104	Mr.Selvam	30	M	7	35	14	Sigmoid volvulus	Resection anastomosis	Discharged
105	Mr.Muniyandi	22	M	1	9	10	Meckeles diverticulum adhesion	Resection anastomosis	Discharged
106	Mrs.Majeema	27	F	1	11	19	Torsion ovarian cyst L	Laparotomy R Oopherectomy	Discharged
107	Mr.Arumugam	60	M	3		32	Small bowel gangrene	Hemicolectomy Ileostomy	Died
108	Mr.Palani	45	M	2		27	Gasrtic perforation	Laparotomy perforation closure	Died

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
109	Mr.Mani	52	M	3		14	Duodenal perforation	Laparotomy perforation closure	Died
110	Mr.Sathish	18	M	3	15	14	Meckeles diverticulum adhesion	Laparotomy resection anastomosis	Discharged
111	Mr.Durai	40	M	1	11	14	Duodenal perforation	Laparotomy perforation closure	Discharged
112	Mr.Chinnapayan	45	M	3	15	10	Intussuception	Resection anastomosis	Discharged
113	Mr.Balaraman	65	M	3		25	Duodenal perforation	Laparotomy perforation closure	Died
114	Mr.Rajasekar	19	M	1	11	16	Duodenal perforation	Laparotomy perforation closure	Discharged
115	Mr.Ganesan	40	M	1	13	20	Ruptured Liver abscess	Peritoneal lavage	Discharged
116	Mrs.Oonchiammal	60	F	2	31	24	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
117	Mrs.Saritha	29	F	1	12	15	Gangrenous appendicitis	Peritoneal lavage & appendicectomy	Discharged
118	Mr.Vinodkumar	38	M	1	11	14	Duodenal perforation	Laparotomy perforation closure	Discharged
119	Mr.Narayanasamy	55	M	3		32	Ileal gangrene	Resection &Jejunostomy cecostomy	Died

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
120	Mr.Raja	22	M	1	10	10	Duodenal perforation	Laparotomy perforation closure	Discharged
121	Mr.Shanmugam	71	M	4		32	Duodenal perforation	Laparotomy perforation closure	Died
122	Mrs.Elavarasi	40	F	1		25	Gangrenous appendicitis	Peritoneal lavage	Died
123	Mrs.Minnalu	35	F	5	11	25	Appendicular abscess	Peritoneal lavage	Discharged
124	Mr.Subramani	46	M	1	13	20	Duodenal perforation	Laparotomy perforation closure	Discharged
125	Mrs.Shanthi	48	F	2	35	25	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged
126	Mr.Vijayakumar	22	M	1	15	14	Duodenal perforation	Laparotomy perforation closure	Discharged
127	Mr.Gnanavel	26	M	1	16	10	Duodenal perforation	Laparotomy perforation closure	Discharged
128	Mrs.Anjalai	39	F	2		36	Hollow viscus perforation with ca cervix	B/L flank drain	Died
129	Mrs.Poonghauli	29	F	4		31	Ceacal volvulus	R Hemicolectomy end to end anastomosis	Died
130	Mr.Raji	50	M	1	11	15	Prepyloric perforation	Laparotomy perforation closure	Discharged

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
131	Mr.Dharmaraj	15	M	1	16	14	Appendicular perforation	Peritoneal lavage & appendicectomy	Wound sepsis & Discharged
132	Mr.Chandra Sathiya	37	M	1	12	14	Duodenal perforation	Laparotomy perforation closure	Discharged
133	Mr.Sridhar	34	M	1	11	14	Duodenal perforation	Laparotomy perforation closure	Discharged
134	Mr.Koteeswari	30	F	1	25	23	Transverse colon gangrene	R Hemicolectomy end to end anastomosis	Discharged
135	Mr.Gopal	60	M	5		31	Duodenal perforation	Laparotomy perforation closure	Died
136	Mr.Murugesan	30	M	1	18	14	Duodenal perforation	Laparotomy perforation closure	Discharged
137	Mrs.Muniammal	30	F	1	15	19	Prepyloric perforation	Laparotomy perforation closure	Discharged
138	Mr.Karthikeyan	34	M	1	9	14	Duodenal perforation	Laparotomy perforation closure	Discharged
139	Mr.Senthilkumar	30	M	1	12	14	Duodenal perforation	Laparotomy perforation closure	Discharged
140	Mr.Adhikesavan	30	M	2	14	20	Gangrenous appendicitis	Peritoneal lavage	Discharged
141	Mrs.Egavalli	50	F	1	15	25	Appendicular perforation	Peritoneal lavage & appendicectomy	Discharged
142	Mr.Madhavan	44	M	3		23	Ruptured Liver abscess	Peritoneal lavage	Died

S.No	Name	Age	Sex	Day of Intervention	No. of days stayed in Hospital	Mannheim Score	Diagnosis	Treatment	Outcome
143	Mr.Saranraj	19	M	1	11	10	Duodenal perforation	Laparotomy perforation closure	Discharged
144	Mr.Murugesan	50	M	3	45	32	Ileal gangrene	Resection anastomosis	Wound sepsis & Discharged
145	Mr.Arumugam	40	M	3		19	Duodenal perforation	Laparotomy perforation closure	Died
146	Mr.Jeeva	20	M	1	43	10	Stab injury	R Hemicolectomy end to end anastomosis	Wound sepsis & Discharged
147	Mr.Veeraragavan	50	M	1	38	32	Internal hernia & Banding with adhesion	Resection anastomosis	Discharged
148	Mr.Arumugam	60	M	1	12	19	Gasrtic perforation	Laparotomy perforation closure	Discharged
149	Mr.Annappan	55	M	1	11	15	Duodenal perforation	Laparotomy perforation closure	Discharged
150	Mr.Govindaswamy	32	M	2	18	14	Duodenal perforation	Laparotomy perforation closure	Wound sepsis & Discharged